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A THERAPEUTIC VIEW OF THE METABOLIC SYNDROME

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Abstract

The article presents the author's own understanding of MS and its components based on extensive personal experience.

Key words: *metabolic syndrome, symptomatic arterial hypertension, metabolic arterial hypertension, insulin resistance, hyperinsulinemia.*

There are a lot of publications in modern domestic and foreign literature devoted to metabolic syndrome (MS). Thematic journals are published, the international institute "Metabolic Syndrome" has been established, conferences, symposiums, and congresses dedicated to MS are held. Almost all Uzbekistan and international conferences, congresses, and congresses pay great attention to this topic, but there is still a paradox: in practical conditions, MS is not diagnosed and, consequently, patients do not receive adequate pathogenetic therapy. The purpose of this article is to present our ideas on this problem and attract the attention of colleagues involved in it, as well as practitioners, to discuss unclear issues both in the field of terminology, understanding the causes and mechanisms of MS development, and in the diagnosis and treatment of MS [1-6]. Let's start with the terminology. Metabolic disorders, combined with the presence of certain clinical symptoms, combined into various syndromes, have long attracted the attention of doctors around the world. So, even the great Hippocrates in the IV century BC described Nabitus apoplexicus. In 1922, F. Lang, when observing patients with arterial hypertension (AH), associated it with obesity, impaired carbohydrate metabolism, and gout. and in 1923, E. Kylin described a syndrome in which hypertension, hyperglycemia and hyperuricemia were combined. Different combinations of specific abdominal obesity, explained by metabolic disorders, with some clinical syndromes received different names. Among them were "metabolic tri syndrome", "polymetabolic syndrome"," abundance syndrome "(N. Mehnert and N.," hormonal metabolic syndrome " and"metabolic vascular syndrome". In 1989, N. coined the term "fatal quartt", which includes hyperlipidemia,as well as impaired carbohydrate tolerance and insulin resistance [6]. The most accepted term for this phenomenon at present is the term "metabolic syndrome", proposed in 1981 by M. Hanefeld, W. Leonardt, although the founder of the theory of MS is considered to be the American scientist G. Reaven, who singled out insulin resistance among the observed metabolic disorders. his Buntigton lecture, delivered in 1988, went down in the history of medicine as "the birth of MS" [1-6]. Since then, the recommendations of various expert commissions on MS (WHO, 1998; US National Committee on Cholesterol, 2001; International Diabetes Federation, 2005) often suggest diagnostic criteria

that differ slightly from each other. For example, experts of the US National Committee on Cholesterol recommended using the following criteria to detect MS in practice[3]: abdominal obesity — waist circumference (OT) in men more than 102 cm and in women more than 88 cm; AD-systolic blood pressure (AD) more than 130 mm Hg and/or diastolic AD more than 85 mm Hg;which is located under the skin, as well as in the omentum and near the mesenteric space. It was studied that the main cells of adipose tissue-adipocytes-can be white and brown and differ only in the number of mitochondria, which give the adipocyte a brown color. Adults have very few brown adipocytes. In the late 80s of the twentieth century, it was discovered that adipose tissue is a place of intense metabolism of sex steroids. Further, a number of studies revealed that adipose tissue is a kind of endocrine gland that secretes a number of hormones and biologically active peptides. These include leptin, pantophysin, resistin, tumor necrosis factor β, adiponectin, visfatin, intra-adipocyte alternative proteins (adipsin, C3, B), intra-adipocyte protein 30 kD (ACRP30), acetylation-stimulating protein (ASP), lipoprotein lipase (LPL), cholesterol ester transfer protein, apolipoprotein e, retinol binding protein, vascular endothelial growth factor (VEGF), interleukin 6, angiotensinogen, plasminogen activator ainhibitor type 1 (PAI-1), transforming growth factor β, hepatocyte growth factor, insulin-like growth factor 1 (IGF-1), monobutyrin, proteins types 1, 2 and 3, uncoupling oxidative stress phosphorylation, synthesis of induced nitric oxide, which increases the level of free fatty acids, insulin resistance and hypertriglyceridemia, prostacyclin (PgI2), acute phase proteins (haptoglobin, α1-acid glycoprotein), extracellular matrix proteins (collagen types 1, 3, 4 and 6, fibronectin, osteonectin, laminin, matrix metalloproteinases types 2 and 9), estrogens (P450 aromatase converts androstenedione to estrone), 17-β-hydroxysteroid oxidoreductase, and some others. It is assumed that adipocytes of the omentum and retroperitoneal space are the most active in the development of MS, as they develop factors that affect the development of the IR phenomenon. Predominant fat deposition under the skin, in the greater omentum and mesentery is characteristic of the abdomen: high levels oftriglycerides (TG) (more than 1.7 mmol/L); low levels of high-density lipoprotein cholesterol (HDL) — less than 1 mmol/L for men and less than 1.3 mmol/L for women; blood glucose levels in the blood are very high. fasting blood plasma of more than 6.1 mmol/ l or 2 hours after an oral glucose tolerance test of more than 7.8 mmol/l. Currently, there is a general understanding of modern MS diagnostic capabilities. The WHO recommendations (2001) are quite specific and easy to apply [6]. Russian experts on the diagnosis and treatment of MS in the recommendations of the Consensus of Russian Experts on the problem of MS in the Russian Federation in 2010 suggested that when considering the possibilities of diagnosing MS, the main feature should be taken into account-central android obesity, in which the OT in women was more than 80 cm, and in men-more than 94 cm, and additional criteria: an increase in the level of TG more than 1.7 mmol/l, low-density lipoprotein cholesterol(LDL) more than 3 mmol/L, a decrease in the concentrationof LDL LDLcholesterol less than 1 mmol/l in in men and less than 1.2 mmol/ l in women, the presence of fasting hyperglycemia with a plasma glucose level of more than 6.1 mmol/l, impaired glucose tolerance with a plasma glucose level of more than 7.8 and less than 11.1 mmol/l 2 hours after taking carbohydrates.1 Blood pressure should be 130 and 80 mm Hg and/or more. Thus, in accordance with Russian recommendations, MS can be diagnosed in the presence of the main trait in combination with two additional criteria, while the mandatory presence of A G is practically not taken into account.We believe that this is incorrect, and suggest that we talk about the presence of hypertension in a MS patientin combination with signs of impaired both lipid and carbohydrate metabolism [6]. This suggestion is based on our observations, which showed a significant increase in OT in the

absence of any deviations in the parameters of lipid and carbohydrate metabolism. At the same time, the addition of AD with an increase in OT was the first sign of MS. Separately, we want to discuss the issue of increased OT, which almost all researchers consider the main feature in the diagnosis of MS. At the same time, there is still no unified standard for measuring OT. Measuring persons choose their waist position at random, which should not be the case. We define FROM as follows. It is necessary to apply a centimeter tape in the middle of the distance between the navel and the lower part of the xiphoid process of the sternum and measure when the patient makes a deep exhalation, combined with the maximum retraction of the abdomen. In this case, OT can be measured in centimeters or inches, but the location of this measurement must be specific andspecific. For what purpose OT is measured As you know, the human body consists of 25-27% adipose tissue, co. Clinical identification of MS (any of the three risk factors) Risk factor Determining the level of Abdominal obesity-FROM, cm: men More than 102 women More than 88 TG, mmol / l More than 1.7 HDL cholesterol, mmol/L: men Less than 1.0 women Less than 1.3 Blood pressure, mmHg: More than or equal to systolic 130 and/or more diastolic 85 and/or more Fasting glucose, mmol/ l More than 6.1 in accordance with the accepted criteria without increasing BMI. At the same time, it should be assumed that obesity and MS are only stages of the same disease. In a person with simple obesity, other signs accumulate over time, and if they are present, the patient must already make a formidable diagnosis of MS. Currently, almost no attention is paid to assessing the severity of MS. We have already informed the medical community about our proposal to assess the severity of MS. We will briefly recall them now. We suggest subdividing MS according to the number of points for each component into mild, moderate and severe MS. To do this, it is necessary to assess the severity of abdominal obesity — according to OT, dislipidemia, the degree of increased arterial hypertension, the degree of carbohydrate metabolism disorders, the manifestations of at hero thrombosis (according to clinical and instrumental data), as well as the severity of hyperinsulinemia. Assessment of the severity of hyperinsulinemia it should be performed according to a special test-questionnaire, which was created by us and published [4, 5]. It was based on subjective symptoms of poor health that occur on an empty stomach, after a meal, and when skipping the next meal. It is assumed that depending on the results of this test, conclusions can be drawn about the presence of hyperinsulinemia, both permanent (basal), and fasting and postprandial. The test questionnaire is completed and evaluated by the patient initially and during treatment. The overall assessment involves evaluating subjective feelings on the following scale: (-) — no symptoms; (+) — mild severity; (++) — moderate severity; (+ + +) — clear severity; (+ + + +) — sharp severity of symptoms. The degree of persistent (basal) hyperinsulinemia was assessed by the severity of such indicators as the speed of food consumption, general irritability, decreased performance, fatigue in the evening, fatigue in the morning, decreased intelligence, memory, forgetfulness, depression, anxiety, sadness. Symptoms of hyperinsulinemia on an empty stomach, during fasting, or when skipping a regular meal were assessed by the severity of excessive sweating, trembling in the hands, palpitations, dizziness, impaired concentration of thought, hunger, headache intensity, sudden mood changes, visual impairment, and bouts of loss of strength. Symptoms of impaired stimulated postprandial insulin secretion were assessed by the presence of episodes of lethargy, weakness, fatigue, drowsiness, impaired concentration of thought, attacks of fog before the eyes, dizziness, sweating after eating. After calculating the total number of signs, they are converted into points on a special scale for assessing the degree of hyperinsulinemia: 1 point-up to 20 signs, 2 points - from the nal (android, central) type of obesity, in which the figure takes the shape of an apple. The predominant deposition of fat in the

lower torso and thighs is characteristic of the female, gynoid type of obesity, in which the figure takes on the shape of a pear. It is assumed that abdominal fat is characterized by reduced sensitivity of adipocyte receptors to the anti-lipolytic effect of insulin and increased sensitivity to the lipolytic effect of catecholamines, as a result of which a large amount of free fatty acids is produced, rushing into the portal vein system and deposited in the liver and pancreas. analysis of the sequence of development of clinical manifestations of MS suggests that abdominal obesity is its earliest clinical symptom. It is believed that the process of including the pathogenetic stages of MS without abdominal obesity is impossible. At the same time, we observed a number of patients who, in the presence of severe abdominal obesity, did not have any manifestations of lipid or carbohydrate metabolism disorders, or even an increase in blood pressure. You can think that the next stage in the development of this disease is hepatosteatosis and fatty degeneration of the pancreas. Currently, neither hepatosteatosisnor pancreatic adipose dystrophy are included in the criteria for the diagnosis of MS. We believe that this issue deserves special study. In the criteria for the diagnosis of MS, there is also no mention of such an indicator as the body mass index (BMI). We believe that when formulating a diagnosis, this indicator should be used to characterize a specific variant of MS. BMI (body massindex — BMI) was developed by the Belgian scientist Adolph. We believe that BMI, like OT, is an important anthropometric parameter that allows differentiating types of diabetes mellitus (DM) and performing dynamic monitoring of the patient and evaluating the effectiveness of treatment measures. BMI is calculated using the formula I = m/h2, where m is body weight, kg; h is height, m. According to WHO recommendations, an interpretation of BMI indicators has been developed, presented in. Since obesity can exist without the presence of MS, MS can also be diagnosed today in terms of leptin secretion and reduce the possibility of dietary control of obesity. The presence of a heterozygous polymorphism in the leptin receptor gene may slightly increase the level of TG and reduce HDL, affecting the predisposition to obesity and type 2 diabetes. The presence of a homozygous polymorphism in the transcription factor PPAR α gene is characterized by impaired lipid and carbohydrate metabolism and increases the likelihood of developing atherosclerosis, CHD, and type 2 diabetes. A favorable factor preventing the development of MS is the presence of a heterozygous polymorphism in the transcription factor gene PPAR γ, which reduces the risk of developing insulin resistance, hyperinsulinemia, and type 2 diabetes. Therapy of metabolic syndrome. Since MS is almost certainly based on insulin resistance, treatment should be focused primarily on its elimination. Treatment should be based on both correction of insulin resistance and maximum reduction of hyperinsulinemia , as well asmeasures to organize an appropriate lifestyle for the patient. Among them, the first place should be dosed physical activity, carried out regularly, at least four times a week, lasting at least 40 minutes, which helps to increase the sensitivity of cell receptors, especially muscle ones, to insulin. This eliminates insulin resistance, rather than burning calories. It is not necessary to visit expensive fitness clubs. The most rational way is to walk at a certain speed (100-150 steps per minute). even more attention should be paid to nutrition. The basic principle of nutrition should be the absence of excessive food stimulation of endogenous insulin secretion, especially in the morning, since morning hyperinsulinemia and morning insulin resistance are most pronounced. You should know that dietary stimulants such as carbohydrates, especially simple ones, provide hyperinsulinemia almost all day long. Dietary measures should not be designed for a temporary course, but for a long period, preferably for life. They should be corrected in a timely manner by improving nutrition, which should be aimed at maximizing the inhibition of excessive stimulation of endogenous insulin production. Evaluation of effectiveness is determined by indicators of hyperinsulinemia according to the

questionnaire test. Frequently offered dietary recommendations that focus on low-fat and 20 to 40 characters, 3 points — from 40 to 60 characters, 4 points - from 60 to 80 characters, 5 points-more than 80 characters. The severity of abdominal-visceral adipose tissue deposition (in points) was assessed by OT (Table 3). The severity of dyslipidemia was assessed by the level of total cholesterol (TC): 5.5—6.5 mmol/l-1 point, 6.5—7.5 mmol/l-2 points, 8.5—9.5 mmol/l-3 points, more than 9.5 mmol/ l — 4 points. any increase in the level of TC in combination with an increase in the level of TG is considered 5 points. In the presence of an unfavorable HDL-LDL ratio, 1 point is added to the assessment of the CCH level. The severity of hypertension was also evaluated in points: grade I hypertension-3 points, grade II hypertension-4 points, grade III hypertension-5 points. Violation of carbohydrate metabolism was evaluated at 1 point when fasting hyperglycemia was detected; violation of glucose tolerance was evaluated at 2 points, type 2 diabetes (on a diet) - at 3 points, type 2 diabetes requiring correction by oral therapy-at 4 points, type 2 diabetes requiring insulin therapy, — 5 points. The severity of at hero thrombosis in the presence of ischemic heart disease (CHD) and tension angina was estimated at 3 points, the presence of unstable angina or myocardial infarction — at 4 points, the combination of CHD (myocardial infarction) with stroke — at 5 points. случаеWhen the total score was 10 or less, the severity of MS was considered mild, from 11 to 20 points-moderate, 20 points or more — severe. The severity of MS over time may change with successful treatment. We believe that patients with MS can already be divided according to clinical manifestations into several subtypes: with hypertriglyceridemia, with leptinresistance, with hyperuricemia, with type 2 diabetes, with a high level of C-reactive protein, which is important for determining a differentiated management strategy for patients. Clinical types of MS can be predicted and confirmed by analyzing the polymorphism of a number of genes. Detection of the presence of a homo - or heterozygous variant in the genes, ghrelin, leptin, leptin receptor, transcription factor PPAR α and transcription factor PPAR γ in the genotype of an individual provides grounds for predicting the course of the disease. Thus, a heterozygous polymorphism in the ectonucleotide pyrophosphatase gene leads to the formation of a substance that blocks the α-subunit of the insulin receptor mainly in the brain and pancreas and interferes with the binding of insulin. The presence of a heterozygous polymorphism in the ghrelin gene is associated with an increased risk of developing MS, which occurs with increased fasting glucose levels, glucose tolerance, and low HDL levels. With the homozygous GG genotype for the INSIG2 gene, the individual is assumed to have more subcutaneous adipose tissue. The presence of a heterozygous polymorphism in the leptin gene may increase the risk of endocrinologists, gastroenterologists, and surgeons working with this group of patients, and, of course, dietitians should have a clear understanding of the independence of the nosological form of MS and the methods of MS therapy, both medical and non-medical. When MS is detected, pathogenetically sound therapy should become a priority for specialists not only in the healthcare system, but also in the food industry, and the Committee on physical Culture and Sports, and, of course, should serve as a reason for creating material motivation for the population to take care of their health. The dynamics of MS correction should be carried out according to anthropometry, a questionnaire test, and the so-called food diary. Prevention of MS should begin in families with a burdened heredity for this pathology. To increase motivation, it is necessary to carry out genetic labeling. The basis of prevention should be psychotherapeutic support — physical activity, training in nutrition according to the daily biological rhythm and determining the compatibility of the products taken. At the same time, it is desirable to select the quantity and quality of food products based on the calculation of daily carbohydrate intake. It is necessary to encourage the

production of products based on natural sweeteners without the use of fructose and glutamates. Fructose intake is carefully analyzed. Recently, dietary recommendations for the use of fructose have become somewhat more moderate, but fructose-based products are becoming more and more common. The use of fructose in the diet of patients with MS leads to a more rapid formation of TG — the main component of adipocytes. For MS, high-calorie (isomaltase, sucrolase) and calorie-free (erythritol, stevia) sweeteners should be recommended. It is possible to determine insulin resistance and the presence of hyperinsulinemia by laboratory methods. At the same time, you can determine the concentration of proinsulin, the level of insulin (both basal and stimulated); 2 and 3 hours after breakfast (125 g of simple carbohydrates), you can determine the HOMA index and insulin resistance. These studies, however, have little use in practical health care. The analysis of hyperinsulinemia symptoms is most acceptable using a questionnaire test, which the patient must complete independently with further monitoring of its dynamics. Despite the fact that no one doubts the existence of MS, in most countries of the world it is not considered as a separate nosological form. Only in the United States is MS recognized as a disease with the identification number ICD-9-CM code 277.7. The lack of a nosological unit significantly complicates the adequate management of patients with MS. Discussions about the nature and variants of MS are still far from complete, but despite this, a convincing justification has already been created for separating MS into a separate nosological form-a metabolic disease. We suggest calling MS a metabolic disease. low-calorie diet, do not correspond to the essence of the pathogenesis of MS. In case of insufficient effectiveness of dietary and physical measures, medical effects should be carried out, also aimed at eliminating insulin resistance and hyperinsulinemia. Currently, one of the drugs of choice for MS pharmacotherapy should be metformin, which belongs to the biguanide group. The drug increases hepatic and peripheral sensitivity to endogenous insulin without stimulating its secretion. Increasing the sensitivity of peripheral tissues, especially myocytes, to the action of insulin under the influence of metformin is realized through a number of cellular mechanisms. Under the influence of metformin, the number and affinity of insulin receptors increase. In addition, the tyrosine kinase activity of insulin receptors is stimulated, as well as the expression and activity of glucose transporters, their translocation from the intracellular pool to the cell membrane. These processes lead to an increase in glucose uptake by the target organs of insulin: the liver, muscle and adipose tissues; the synthesis of glycogen in the liver increases.

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