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METABOLIC SYNDROME: DOCTOR'S TACTICS

Abstract:
The article presents the main criteria for diagnosis, clinical and pathogenetic characteristics of metabolic syndrome, as well as the doctor's tactics for correcting the conditions combined by the term metabolic syndrome, and measures to prevent the further development of related diseases.

Key words*: metabolic syndrome, diagnosis, doctor's tactics, prevention.*

The most frequent combination of nosologies and conditions in patients after 40 years is a combination of diabetes, obesity, arterial hypertension, hypercholesterolemia, hyperuricemia, although this combination has become significantly "younger" in recent years. All these conditions were proposed to be united by the term "metabolic syndrome". For the first time J.P. Camus in 1966 proposed the term "metabolic trisyndrome", and in 1980 M. Henefeld and W. Leonhardt modified this concept into "metabolic syndrome".

Most modern doctors and scientists are guided by criteria for defining metabolic syndrome (MS), according to which MS includes impaired glucose tolerance or type 2 diabetes and insulin resistance combined with two or more of the following criteria: an increase in blood pressure (BP) up to 160/ 90 mm Hg art.; an increase in the level of triglycerides (TG) in the blood plasma (more than 1.7 mmol/l) and/or a low level of high-density lipoprotein cholesterol (HDL) less than 0.9 mmol/l in men and less than 1.0 mmol/l in women; abdominal obesity (the ratio of waist circumference (WC) to hip circumference (HC): more than 0.9 in men and more than 0.85 in women; and/or excess body mass index (BMI) above 30 kg/m²); microalbuminuria (albumin content in urine 30 mg/dL or ratio of albumin to creatinine 30 mg/g). The International Diabetes Federation (IDF) proposed slightly modified criteria, where abdominal obesity comes to the fore, and according to the IDF, the presence of two or more risk factors is mandatory: TG level above 1.7 mmol/l ; level of HDL cholesterol <1.03 mmol/l in men and <1.25 mmol/l in women or the fact of antilipidemic therapy; Blood pressure above 130/85 mm Hg. Art. or the fact of antihypertensive therapy; fasting capillary blood glucose is above 5.6 mmol/l. However, according to the recommendations of American cardiologists and lipidologists, it is proposed to consider the determination of fasting venous blood plasma glycemia above 6.1 mmol/l or impaired glucose tolerance with glycemia above 11.1 mmol/l 2 hours after a glucose load as criteria for MS. Abdominal obesity is at the heart of this condition, which, in turn, can be caused by genetic predisposition, excessive secretion of neuropeptide Y in the arcuate nucleus of the hypothalamus, with hypothalamic syndrome; Leptin resistance is observed in patients with sclerosed ovarian syndrome. Increased appetite, hyperphagia, and decreased sensitivity to leptin are often caused by other neuropeptides, such as endocannabinoids, which are synthesized from arachidonic acid and modulate retrograde suppression of neurotransmitter release; cocaine- and amphetamine-regulating transmitter. With abdominal obesity, adipose tissue is mainly localized in

the peritoneum, extraperitoneally, in the mesentery, and periportally. Visceral adipose tissue includes white adipocytes, which on their surface contain a large number of β-adrenergic, corticosteroid and androgen receptors, while the number of β-adrenergic and insulin receptors is significantly reduced compared to other tissues of the body. Adipocytes synthesize pro-inflammatory cytokines, reduce the fibrolytic properties of blood, and increase the concentration of leptin in the blood. Leptin is a hormonal peptide that regulates the processes of appetite balance and thermogenesis. Leptin is able to activate the sympathoadrenal system, which explains the occurrence of hypertension in obesity. When the sympathoadrenal system is activated, the above-mentioned white adipocyte receptors are stimulated, which, in turn, stimulates lipolysis, lipid peroxidation, and a significant amount of free fatty acids and free radicals are released into the bloodstream. Free fatty acids, entering through v. portae in the liver, reduce its absorption of insulin, which leads to hyperinsulinemia. By reducing the activity of phosphatidylinosinone-3-kinase of insulin receptors, free fatty acids inhibit the transport of glucose into peripheral cells (these mechanisms can explain the occurrence of insulin resistance in peripheral tissues). In parallel with the above mechanisms, free fatty acids activate gluconeogenesis, which leads to an increase in the level of glycemia, and TG and very low-density lipoprotein cholesterol (VLDL-C) are also synthesized from them. Insulin resistance of peripheral tissues is the basis of the pathogenesis of hypertension in MS. In addition to the specified pathogenetic mechanisms, adipose tissue increases metabolic needs, which leads to an increase in heart rate (HR) and cardiac stroke volume, which, in turn, contributes to the development of eccentric hypertrophy of the left ventricle and the formation of diastolic dysfunction, the so-called "obesity cardiomyopathy ". So, guided by such criteria as abdominal obesity and two of the following risk factors: blood pressure above 140/90 mm Hg. art.; reduced content of high-density lipoprotein cholesterol; increased level of triglycerides; microalbuminuria; insulin resistance, the doctor can establish "metabolic syndrome".

Taking into account the pathogenetic features of MS, the recommendations of the American Diabetes Association (2015) and the recommendations of the Unified Protocol for the provision of medical care for type 2 diabetes mellitus, treatment and prevention measures should begin with lifestyle correction. First of all, the doctor must convince his patient to give up smoking, monitor tobacco dependence at every appointment, justify his intentions with the increased risk of cardiovascular disasters when using tobacco. People who consume alcohol should be talked to about how to minimize it. Correcting the nutrition of patients with MS is the key to successful treatment. The attention of patients should be drawn to the fact that their diet should be aimed not only at reducing glycemia, but also at reducing body weight. It is necessary to offer the patient to keep a diary to count carbohydrates or their caloric content in food or to determine the number of bread units. Check this diary every chance you get and point out mistakes, make recommendations for corrections if you find them, and constantly encourage him to lose weight. Convince the patient that a 5-10% loss of body weight significantly reduces the IR of the body. The diet for such patients should contain: a low level of carbohydrates, less than 7% of the total caloric content - saturated fats, dietary fiber and products containing whole grains - half of the diet. Food supplements containing antioxidants, vitamins E, C, carotene, chromium are not recommended, as their effectiveness has not been confirmed. It should be remembered that such patients need to periodically monitor the lipid profile, the functional state of the kidneys, and the consumption of proteins. Physical activity in patients with MS contributes to the reduction of mortality from cardiovascular diseases, the level of IR, and helps prevent the development of diabetes in obese people. Physical activity of at least 150 minutes a week should be moderate. Loads should be regular, in the absence of contraindications, strength exercises are allowed no more than 3 times a week. A detailed and individualized approach to lifestyle correction is the key to successful treatment, prognosis and quality of life of patients. When conducting drug therapy, the multifaceted nature of this condition should also be taken into account. Metformin (diaformin) should be prescribed in order to eliminate IR in MS and to prevent the development of diabetes, since its main effect is to reduce the resistance of peripheral tissues to their own insulin. The mechanism of action of metformin is related to extrapancreatic action and does not affect the secretion of insulin by b-cells of the pancreas; reduces the absorption of carbohydrates in the intestines; increases the conversion of glucose to lactate in the gastrointestinal tract; strengthens the binding of insulin to receptors; increases the transport of glucose through the membrane into the muscles; reduces gluconeogenesis; lowers the level of triglycerides and low-density lipoprotein cholesterol (LDL cholesterol); increases the level of high-density lipoprotein cholesterol (HDL-C). Metformin has a low risk of hypoglycemia, is able to contribute to the normalization of body weight, and has a cardioprotective effect. The prescribed prophylactic dose of metformin is 500–850 mg 2 times a day. It has been proven that metformin at a dose of 850 mg 2 times a day for 3 years reduces the risk of developing diabetes by 31% compared to placebo. The use of metformin in patients under 45 years of age and in patients with severe obesity (BMI >35) reduces the risk of developing diabetes by 44–53%. The only limitation of metformin use is its gastrointestinal side effects (nausea and diarrhea), which may occur in the first two weeks of its use. Prolonged forms of the drug reduce the frequency of side effects, for example, diaformin SR, which causes 2 times less side effects compared to other metformins. Diaformin SR has a dosage of 1000 mg in 1 tablet, which allows it to be prescribed once a day, including for preventive purposes. An increase in blood pressure in such patients is corrected by using angiotensin-converting enzyme inhibitors, in case of their intolerance - angiotensin II receptor blockers. If necessary, to achieve target blood pressure levels (less than 140/90 mm Hg), it is possible to use thiazide (if GFR ≥30 ml/min per 1.73 m²) and

loop diuretics (if GFR <30 ml/min per 1 .73 m²). An important component of therapy is hypolipidemic therapy with statins and antiplatelet therapy. Many studies focus their attention on oxidant stress, which is reflected in the pathogenesis of this condition. Therefore, it is recommended to add drugs that inhibit lipid peroxidation and block free radicals (α-lipoic acid and meldonium) to the main treatment. Therefore, when treating an overweight patient, the doctor must: determine the degree of obesity (according to BMI) and its type; detect IR (according to the NOMA index, carrying out a glucose tolerance test with exercise); measure blood pressure every time a patient visits; send to determine the lipid profile and the level of uric acid in the blood; prescribe lifestyle correction and preventive treatment. When establishing a diagnosis, the doctor must convince the patient that together they are a team in the fight against the disease, help him realize that giving up bad habits, losing weight, eating healthy, and increasing physical activity will help not only to prolong, but also to improve the quality of his life. And only if necessary, the selection of adequate medicinal products in prophylactic or therapeutic doses - metformin (Diaformin SR), antihypertensive drugs, statins - will reduce the risk of MS-related diseases and their complications.

Conclusions. Therefore, the combination of all MS nosological units into a single syndrome enables the doctor to identify metabolic disorders in such patients, together with them to predict risks and determine an individual program for life correction and treatment for the future.

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ANEMIA AND COVID-19

Relevance.

Hematological changes such as thrombocytopenia, leukopenia, lymphopenia, increased neutrophil count, hypercoagulability and elevated D-dimer levels are common features of coronavirus infection (COVID-19), especially in severe cases [1].

Anemia is often associated with chronic diseases of patients. It has been found to be a factor in increasing the risk of mortality and hospitalization in comorbidities such as heart failure, chronic obstructive pulmonary disease and myocardial infarction [2], [3], [4].

Objective. The aim of this review is to analyze studies on the impact of COVID-19 on the development and course of anemia in hospitalized patients.

Discussion. A study conducted by Gaetano Bergamaschi and colleagues from the San Matteo Hospital Foundation investigated the prevalence, pathogenesis and clinical significance of anemia among 206 patients with COVID-19 at the time of their admission to the general medical unit. According to their results, the prevalence of anemia in patients with COVID-19 was 61%, compared to 45% in a control group of 71 patients who had clinical and laboratory findings suggestive of COVID-19, but nasopharyngeal swab tests negative for SARS-CoV-2 RNA (p=0.022). Mortality was higher in patients positive for SARS-CoV-2.

In COVID-19, women had lower hemoglobin concentration than men and a higher prevalence of moderate and severe anemia (25% vs. 13%, p=0.032). In most cases, anemia was mild and caused by inflammation, sometimes associated with iron and/or vitamin deficiency. Factors that influenced the