METABOLIC SYNDROME IS A BIG THREAT!

Kuchkorov Sh.B., Kuddusov M.M., Khakimov S.S.
Students of the Samarkand State Medical University, Samarkand, Uzbekistan.
oxygenium1700@gmail.com

Abstract. *Metabolic syndrome is a complex of metabolic, hormonal and clinical disorders that are risk factors for the development of cardiovascular diseases, which are based on insulin resistance and compensatory hyperinsulinemia. The main mechanisms of the effects of chronic hyperinsulinemia on blood pressure are given, and the main symptoms and manifestations of the metabolic syndrome are given. The most common variant of dyslipidemia in the metabolic syndrome is the lipid triad.*

Keywords: *lecture, children; Adults metabolic syndrome; dyslipidemia; insulin resistance; obesity; atherosclerosis; type 2 diabetes; arterial hypertension.*

In recent years, much attention of specialists in various fields of medicine has been attracted by the problem of the prevalence of a symptom complex called metabolic syndrome (MS).

MS is a cluster of metabolic and hormonal disorders united by a common pathophysiological mechanism - insulin resistance (IR) and including impaired glucose tolerance, atherogenic dyslipidemia, arterial hypertension (AH), which are combined with the abdominal-visceral type of obesity. The urgency of the problem is due to both the increase in prevalence and its social significance, since the characteristics combined in the MS significantly affect the life expectancy and mortality of the young population around the world. The frequency of detection of MS among adolescents increased from 4.2% to 7.0%. According to epidemiological studies conducted in our country, MS is diagnosed in half of obese adolescent

children. In industrialized countries, among the population older than 30 years, the incidence of MS is 15-25%. This disease is more common in men than women, and the likelihood of developing it increases with age. In women, the incidence of MS increases in the postmenopausal period.

Almost all components of MS are established risk factors (RFs) for the development of CVD. The mortality of patients with MS from coronary artery disease is 2–4 times higher than in the general population. MS can be complete, when IR is manifested by all of the above disorders, or incomplete, when IR is not combined with all the components of the syndrome.

There are three types of IR depending on the level of violations.

The study of genetic factors that determine the development of IR made it possible to establish its polygenic nature. In the development of insulin sensitivity disorders, the importance of mutations in the insulin receptor substrate genes, glycogen synthetase, hormone-sensitive lipase, β-adrenergic receptors, tumor necrosis factor, as well as molecular defects in insulin signaling proteins has been shown. Currently, IR is more associated with impaired insulin action at the post-receptor level, in particular, with a significant decrease in the membrane concentration of specific glucose transporters (GLUT-4, GLUT-2, GLUT-1). However, in order for existing genetic defects to manifest themselves clinically, acquired changes are also of no small importance, the most significant of which are excessive nutrition and reduced physical activity. IR leads to a gradual depletion of the compensatory capabilities of β-cells, which are probably genetically predetermined (defect of glucokinase and GLUT-2). β-cell dysfunction develops as a result of the combined action of several factors, one of which is glucose toxicity, i.e. a state of prolonged chronic hyperglycemia, which causes a decrease in the secretory response of β-cells to stimulation of an increased concentration of glucose in the blood. The inability of β-cells to provide the required level of insulin hypersecretion leads to the development of carbohydrate metabolism disorders: from a moderate

increase in the concentration of glucose in the blood plasma after a food load, then on an empty stomach to DM 2.

Abdominal-visceral obesity, as a rule, is the first manifestation of MS, one of the key moments in the development and progression of IR and related metabolic disorders. Excessive deposition of visceral adipose tissue is associated with an atherogenic lipid profile, hyperuricemia, and changes in the blood coagulation system. Disorders united within the framework of MS are asymptomatic for a long time and often begin to form in adolescence and adolescence, long before the clinical manifestation of DM 2, AH and atherosclerotic vascular lesions. Excessive deposition of fat in the abdominal region is a prognostically unfavorable factor. Adipocytes of this area are more lipolytically sensitive than adipocytes of other areas of the body and have the most mobile system for releasing free fatty acids into the blood plasma, an increase in the level of which in the portal and systemic circulation causes a number of disorders of carbohydrate and fat metabolism, as well as changes in the fibrinolysis and endothelial functions.

Identification of the visceral type of obesity is most effectively carried out using computed and magnetic resonance imaging. A clear correlation has been established between the degree of development of visceral adipose tissue and waist circumference. Waist circumference of more than 100 cm at the age of 40 years and more than 90 cm at the age of 40–60 years in both men and women is an indicator of visceral obesity. Visceral adipose tissue, in contrast to adipose tissue of other localization, is richer innervated, has a wider network of capillaries and directly communicates with the portal system. Visceral adipocytes have a high density of β-adrenergic receptors, corticosteroid and androgen receptors. These features determine the high sensitivity of visceral adipose tissue to the lipolytic action of catecholamines and the low sensitivity to the antilipolytic action of insulin.

Among the many mechanisms linking abdominal obesity with a high risk of CVD, a special place belongs to lipid metabolism disorders. The main characteristics of dyslipidemia in MS are hypertriglyceridemia, a decrease in HDL cholesterol

levels, an increase in the content of small dense particles of low-density lipoprotein (LDL), a pronounced postprandial rise and long-term persistence of postprandial lipemia (hypertriglyceridemia, an increase in the number of chylomicron remnants, a level of very low density lipoprotein, an increase in apoB levels , Lp(a), LDL cholesterol, free fatty acids. Hypertriglyceridemia is considered a key determinant of IR-related lipid disorders.

Considering the great importance of the occurrence of dyslipidemia in the development of MS, the diagnosis of these disorders should be taken into account, first of all, along with other diagnostic indicators.

Correction of impaired lipid metabolism in patients with MS is an essential part of the secondary prevention strategy. Patients with dyslipidemia that cannot be corrected by diet therapy are prescribed lipid-lowering drugs.

AH is one of the most frequent manifestations of MS. Its pathogenesis in MS is considered as a sequence of hemodynamic, hormonal and cellular disorders. It includes IR, hyperinsulinemia, increased activity of the sympathetic nervous system and the renin-angiotensin system, stimulation of left ventricular hypertrophy and arterial walls, increased sodium reabsorption and blood volume, and increased peripheral vascular resistance. All these effects together contribute to an increase in blood pressure (BP). BP control in patients with MS is essential to improve prognosis. The tactics of managing patients with hypertension and MS has a number of features: 1) immediate initiation of treatment with antihypertensive drugs in combination with non-drug measures; 2) focus on achieving optimal or normal blood pressure - below 130/85 mm Hg. Art., since it is proved that the stabilization of blood pressure at this level and below gives a real organoprotective effect; 3) more frequent use of combinations of antihypertensive drugs, due to greater resistance to lowering elevated blood pressure in patients.

In recent years, a large number of studies have been carried out that have revealed significant changes in hemorheological parameters in patients with MS. A

tendency to a pre-thrombotic state was established, due to an increase in coagulation factors, inhibition of the fibrinolytic system, and a decrease in the antithrombotic potential of the vascular wall. Among the biochemical changes in the coagulation cascade, an increase in the content of fibrinogen, coagulation factor VII, and an increase in the activity of an inhibitor of tissue plasminogen activator are most consistently detected. Changes in the functional activity of platelets are manifested by an increase in their aggregation and adhesive ability, as well as an increase in the reaction of the release of biologically active substances (thromboxane A2 and platelet growth factor).

Thus, the etiopathogenesis of MS is currently complex and not fully understood. The high frequency of atherogenic, diabetogenic, thrombogenic complications in adolescence allows us to consider this symptom complex as an important pediatric problem. Its decoding requires a comprehensive approach involving specialists from various fields of medicine. Only the joint activity of cardiologists, endocrinologists, gastroenterologists, general practitioners will make it possible to fully study the main pathogenetic mechanisms of the formation of MS, to identify the spectrum of clinical manifestations, while focusing on earlier symptoms that are predictors of its development in children. This will allow developing methods for targeted prevention of cardiovascular disease and type 2 diabetes mellitus, diseases of the hepatobiliary and reproductive systems, and thereby reduce the risk of early disability and premature death.

Conclusion. Preventive or therapeutic measures of MS should be aimed at the totality of risk factors, and the goal of treatment is to reduce the overall risk of cardiovascular morbidity and mortality as much as possible. Rational use of available therapeutic options aimed at reducing body weight, normalizing blood pressure, adequate control of glycemia and lipidemia, can significantly improve the prognosis of patients with a high risk of complications.

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