METABOLIC SYNDROME IN CHILDREN AND ADOLESCENTS

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Annotatsiya:
Metabolic syndrome (synonyms: metabolic syndrome X, Reaven syndrome, insulin resistance syndrome) is an increase in visceral fat mass, decreased sensitivity of peripheral tissues to insulin and hyperinsulinemia, which disrupt carbohydrate, lipid, purine metabolism, and also cause arterial hypertension.

In 1988, Professor G. Reaven, in his Banting lecture, based on his own observations and generalization of studies of other authors, put forward a hypothesis according to which insulin resistance, abdominal obesity, arterial hypertension (AH), atherogenic dyslipidemia and coronary heart disease (CHD) serve as a manifestation of the pathological condition, which he proposed to call "syndrome X". In 1989, D. Kaplan introduced the term "deadly quartet": a combination of obesity, hypertension, impaired glucose tolerance and hypertriglyceridemia.

Metabolic syndrome is one of the most pressing problems of modern medicine associated with leading an unhealthy lifestyle. The concept of "healthy lifestyle" includes a balanced diet, maintaining a normal body weight, regular and age-appropriate physical activity, healthy sleep, and avoiding alcohol and smoking.

According to H. Arnesen (1992), metabolic syndrome is understood as a combination of at least two of five disorders:

1. Insulin resistance with reduced carbohydrate tolerance and hyperinsulinemia;
2.Dyslipoproteinemia with hypertriglyceridemia and lowered high density lipoprotein cholesterol;
3. The tendency to thrombosis and an increase in the blood plasma level of the inhibitor of the plasminogen activator;
4. Arterial hypertension against the background of increased activity of the sympathetic nervous system;
5. Generalized obesity with increased secretion of free fatty acids into the portal vein.

The risks of developing metabolic syndrome include:

1. Obstructive sleep apnea syndrome
2. Non-alcoholic steatohepatitis
3. Chronic renal pathology
4. Polycystic ovary syndrome (for women)
5. Low plasma testosterone levels, erectile dysfunction, or both (for men)

Obesity is an independent risk factor for cardiovascular disease with high mortality. There are two types of it: android and gynoid.

Android - manifested by an uneven distribution of fat with excess deposition in the upper half of the body, on the abdomen and an increase in the amount of visceral (internal) fat. There is little fat on the limbs and buttocks. This obesity is called abdominal. The android type of obesity is the main risk factor for the development of arterial hypertension, atherosclerosis and type 2 diabetes mellitus. In visceral (internal) obesity, an excess amount of free fatty acids enters the bloodstream through the portal vein system (an increase of 20-30 times compared with the norm). As a result, the liver is exposed to a powerful and constant exposure to free fatty acids, which leads to a number of metabolic disorders (hyperglycemia, an increase in low density lipoproteins enriched in triglycerides, insulin resistance, hyperinsulinemia). Insulin resistance and hyperinsulinemia contribute to the development of arterial hypertension.

As shown by Dr. Kalle Suoula and colleagues (University of Tampere, Finland), metabolic syndrome significantly correlates with increased stiffness of the arterial wall, which was diagnosed by measuring the pulse wave velocity (PWV). According to the regression analysis, blood pressure, age, waist circumference, and fasting glucose levels independently predicted an increase in arterial stiffness in middle-aged and older people. Thus, this study has demonstrated for the first time that metabolic syndrome is significantly associated with an increase in the PWV pulse wave velocity.

The development of metabolic syndrome depends on the distribution of fat as well as its amount. Excess fat in the abdomen (aka apple shape), especially when it results in a high waist-to-thigh ratio (reflecting a rather low muscle-to-body fat ratio), increases the risk. The syndrome is less common among people who have excess subcutaneous fat around the thighs (called a pear shape) and a low waist-to-hip ratio (reflecting a high muscle-to-body fat ratio).

Excess abdominal fat leads to excess free fatty acids in the portal vein, increasing the accumulation of fat in the liver. Fat also builds up in muscle cells. Insulin resistance develops in combination with hyperinsulinemia. Glucose metabolism worsens, dyslipidemia and arterial hypertension develop. Serum uric acid levels are generally elevated (increasing the risk of gout), with a prothrombotic state (with elevated levels of fibrinogen and plasminogen activator inhibitor I) and chronic inflammation.

Etiopathogenesis

According to modern concepts, the unifying basis of all manifestations of metabolic syndrome is primary insulin resistance and concomitant hyperinsulinemia. Hyperinsulinemia is, on the one hand, compensatory, that is, necessary to overcome insulin resistance and maintain normal glucose transport into the cell, on the other hand, it is a pathological factor that contributes to the emergence and development of metabolic, hemodynamic and organ disorders, which ultimately lead to the development of type 2 diabetes, dyslipidemia.

Until now, all possible causes and mechanisms of the development of insulin resistance in abdominal obesity have not been fully studied: not all components of the metabolic syndrome can be associated and explained only by insulin resistance. Insulin resistance is a decrease in the response of insulin-sensitive tissues to insulin when it is sufficiently concentrated. The study of the factors causing the development of insulin resistance revealed a pronounced genetic predisposition. Of the external factors that adversely affect the sensitivity of tissues to insulin, physical inactivity and excess fat intake are of the greatest importance.

An important role in the development and progression of insulin resistance and associated metabolic disorders is played by the accumulation of excess adipose tissue in the abdominal region, neurohormonal disorders accompanying obesity, and increased activity of the sympathetic nervous system. The use of computed tomography and magnetic resonance imaging made it possible to study the topography of the accumulation of adipose tissue in the abdominal

region and divide it into a visceral (intra-abdominal) form and a subcutaneous one. Studies have shown that a significant increase in the mass of visceral adipose tissue, as a rule, is combined with metabolic disorders, and primarily with insulin resistance. At the same time, a close correlation was established between the degree of development of visceral adipose tissue and the size of the waist circumference (OT).

As the mass of fat in the body increases, the size of the fat cells increases. The larger the adipocyte, the less insulin sensitive it is. Accordingly, the inhibitory effect of this hormone on lipolysis processes is also less. Intense lipolysis in visceral adipocytes leads to the release of large amounts of free fatty acids (FFA), mainly into the portal circulation and the liver. In the liver, FFA interfere with the binding of insulin by hepatocytes, causing the development of insulin resistance at the liver level, a decrease in the extraction of insulin by the liver and the development of hyperinsulinemia. In turn, hyperinsulinemia through impaired autoregulation of insulin receptors enhances peripheral insulin resistance. In muscle tissue, FFA interfere with the utilization of glucose by myocytes, which also contributes to the development of hyperglycemia and compensatory hyperinsulinemia.

Adipose tissue has an auto-, para- and endocrine function and secretes a large amount of substances with various biological effects. Among them, tumor necrosis factor-a (TNF-a) and leptin are found. Many consider TNF-a as a mediator of insulin resistance in obesity. Leptin, secreted primarily by adipocytes, acts at the level of the hypothalamus, regulating feeding behavior and the activity of the sympathetic nervous system, as well as a number of neuroendocrine functions. Studies have shown a BMI-independent positive correlation between leptin production, hyperinsulinemia, and insulin resistance.

In pediatric practice, obesity is subdivided into degrees depending on excess body weight. Diagnosis of obesity is based on measuring body weight, comparing it with the average tabular indicator for a young child, gender and height, and calculating (in%) its excess. At the same time, the degrees of obesity are distinguished: I degree - 10-25%, II degree - 26-49%, III degree - 50-99%, IV degree - 100% or more excess body weight. In older schoolchildren and adolescents, the Quetelet index (body mass index (BMI) = weight (kg) / height (m) 2) can be calculated to determine the degree of obesity.

For example: height - 1.5 m, weight - 48 kg; BMI = 48 kg / (1.5 m) 2 = 21.3 kg / m2.

Diagnostics:

1. Waist circumference and blood pressure
2. Plasma glucose and lipid profile

In accordance with the indicated WHO recommendations, the following fasting plasma glucose levels are of diagnostic value:

1) normal fasting plasma glucose is up to 6.1 mmol / l (<110 mg / dl);
2) fasting plasma glucose from> 6.1 (> 110 mg / dl) to <7.0 mmol / l (<126 mg / dl) is defined as impaired fasting glycemia;
3) a fasting plasma glucose level> 7.0 (> 126 mg / dl) is regarded as a preliminary diagnosis of diabetes mellitus, which should be confirmed by re-determination of blood glucose on other days.

The complex of treatment of metabolic syndrome includes the following equivalent positions: lifestyle change, obesity treatment, treatment of carbohydrate metabolism disorders, arterial hypertension, dyslipidemia treatment.

Changing the lifestyle of a child and adolescent means the following:

- 1. diet and regimen (principles of nutrition);
- 2. physical activity;
- 3. psychotherapy;
- 4. problem-oriented training and self-control. Principles of nutrition for children and adolescents with obesity

In most cases, the main cause of obesity, especially in children, is overeating.

It is necessary to limit the "carbohydrate" height to 150 g per day. It is recommended to consume as little as possible readily soluble and rapidly absorbing carbohydrates. It is necessary to replace them with polysaccharides. The daily dose of easily soluble carbohydrates should not exceed 25-30 g. When drawing up a diet, it is imperative to take into account the glycemic index of foods. The less the product has "ability" to increase the level of glucose in the blood, the more favorable its effect on the insular apparatus and the lower the risk of additional utilization of glucose in fat depots. And foods with a high glycemic index increase this risk. However, it should be borne in mind that in obese patients, almost all carbohydrate-containing foods cause a greater rise in glucose levels than in people of normal weight.

Physical activity in terms of its importance in the prevention and treatment of excess weight is rightfully ranked second after nutrition. Therefore, recommendations for their use are no less than diets.

Before starting physical activity, you need to have a complete picture of your child's health and determine the initial level of fitness. For training the cardiovascular system, walking, playing sports, swimming, cycling, skiing and skating (including roller skates) are well suited. You can and should move anytime and anywhere: sitting in the bathroom, watching TV, on the bus, at a school desk ... You have to walk, run, swim, ride a bike, do exercises, do shaping, etc., etc. It should be remembered that physical activity without diets is ineffective.

The leading treatment for obesity is a combination of diet and exercise. Both diet and exercise require careful, well thought out and strictly individual dosage. But often, when a doctor gives recommendations to a child and his parents, the recommendations to lose weight look like nothing more than wishes. It is not fully understood that the treatment of obesity, like, by the way, the treatment of any other chronic disease, must be continuous. That is, a set of measures aimed at actively reducing excess body weight should in no case end with the child's return to the usual diet and lifestyle for him and his family. It should smoothly transition into a set of measures aimed at maintaining the achieved result (Saris W., et. Al., 2000). This is the approach that a weight loss and weight management education program for obese children and adolescents should support.

First of all, treatment should be aimed at solving the problem of excess weight, which is achieved mainly by non-drug methods. It is necessary to increase physical activity, reduce the caloric content of food and rationalize the composition of food consumed.

In cases of unbalanced nutrition, the food ration can be supplemented with fortified vitamins, trace elements, food additives made on the basis of dietary fiber. If necessary, special drugs are used and, in extreme cases, surgery (gastric or biliopancreatic bypass).

The amount of easily digestible carbohydrates in the diet should be less than 30% of the total caloric content of food, fats - less than 10%.

Thus, considering metabolic syndrome as an independent "generalized cardiovascular-metabolic disease", we propose to focus on the development of unified diagnostic criteria for this condition in children and adolescents. It is necessary to include the diagnosis "metabolic syndrome" in medical standards, the International Classification of Diseases (ICD). Today in ICD 10 there is no code for metabolic syndrome as a nosological form. Some of its clinical markers are encrypted in different headings: type 2 diabetes mellitus - E11; abdominal obesity - E 66.0, etc. At the same time, in adolescents, the metabolic syndrome most often proceeds under the guise of hypothalamic syndrome of puberty and is coded as E 23.3.

From the point of view of evidence-based medicine, it is desirable to conduct targeted multicenter studies of drugs used to treat metabolic syndrome in children and adolescents.

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