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OBESITY AS A METABOLIC FACTOR OF THE RISK OF CARDIOVASCULAR DISEASES

ABSTRACT

Obesity is currently one of the serious problems of modern society and medicine. The article gives an overview of obesity as one of the main metabolic risk factors for cardiovascular diseases. The reasons of the development of obesity and pathogenetic mechanisms of complications development from the side of the cardiovascular system are considered against its background, as well as the risk to human health in the presence of visceral obesity. We present data on child obesity as a metabolic base of cardiovascular diseases at which a prolonged accumulation of adipose tissue leads to the most serious disorders of human organs and systems, primarily cardiovascular one.

Keywords: obesity, overweight, cardiovascular diseases, epicardial fat.

According to the WHO, overweight and obesity are determined as «abnormal or excessive fat accumulation that presents a risk to health'' [5]. Obesity plays a role in the development of a number of cardiovascular risk factors (FR). The researchers defined pathogenetic basis of the negative effect of obesity on the structural and functional activity of heart and blood vessels. An obese person has a greater risk of developing cardiovascular disease (CVD), which in turn can lead to severe heart diseases [24].

Many factors can cause obesity, including genetic (more than 50 candidate genes). But most people do not have monogenic inheritance of obesity. There are many studies being held to find candidate genes that can affect obesity and overweight. Currently, the role of mutations in the PPAR genes (peroxisome proliferator-activated receptors), fatty acid-binding protein 2, (FABP2), ADRB2 and ADRB3 (G-protein adrenergic receptors) is under study. The latter are considered as an interesting finding in nutrigenetics, confirming the hypothesis of «economical genotype». In people with a body overweight and with a mutation of the ADRB3 gene, daily energy consumption, altered lipolysis and increased abdominal obesity decrease are noted [6]. Despite the revealed interrelationships of gene mutations with overweight, the question of direct role of genetic factors in the development of obesity remains controversial. One cannot ignore environmental factors, such as lifestyle, diet, physical activity, stressful situations and bad habits. To date, according to the WHO, the main reasons of obesity are an excessive supply of nutrients with food and a low level of physical activity, which does not allow consuming the amount of energy coming from food [5, 8, 9].

Clinically obesity can be an independent disease (exogenous-constitutional obesity) or a syndrome that develops at various diseases, such as hypothyroidism, hypercorticism, polycystic ovary syndrome, Cushing's syndrome, etc. (in the latter case, excess weight can be eliminated after curing or compensation of the main disease). In this case, it must be remembered that obesity itself leads to disruption of the sexual glands, the hypothalamo-pituitary system and the adrenal glands. For example, the establishment of a diagnosis of neuroendocrine form of the hypothalamic syndrome is inappropriate, since the formation of hypothalamic stigmas such as cyanotic striae, pigmentation in places of friction, the formation of acanthosis of obese, unclean skin and functional disorders of the hypothalamic-pituitary system is not the cause, but the result and manifestation of obesity, and the degree of their severity correlates with the duration and severity of obesity [20].

Excess body weight contributes to increased levels of total cholesterol and low-density lipoprotein (LDL) and very low density (VLDL) in plasma. It has been established that the production of cholesterol (Ch) in people with obesity increases by an average of 20 mg per

each excess kilogram of fat. For every 4.5 kg of body weight, systolic blood pressure (SBP) is increased by 4.5 mm Hg [4, 19, 20].

Obesity occurs when the number or size of fat cells in the body increases. Normally, a person has 30 to 35 billion fat cells. When a person gets fat, these fat cells first increase in size, and then their number grows. When a person grows thin, the size of the cells decreases, and no matter how thin a person is, the number of fat cells remains the same. Namely for this reason, after gaining of fat mass it is very difficult to lose weight.

Dangerous to human health is visceral obesity (abdominal type of obesity), at which the adipose tissue (AT), has an uneven distribution with excess deposition in the region of the upper body and abdomen (intraabdominal visceral fat - in the omentum, mesentery, retroperitoneal area), as well as in internal organs, including the heart, kidneys and liver (extra-abdominal deposits of visceral fat). Just deposits of adipose tissue in vital organs lead to complications and high mortality. It all starts with a violation of the functional activity of AT, which is not only a depot of fat, but an active endocrine and paracrine organ. Visceral fat, being hormonally active, performs a complex role in the body: secretes estrogens (adipocytes aromatase promotes the synthesis of estrogens from adrenal androgens), angiotensinogen, prostaglandins, tumor necrosis factor α, interleukin-6, leptin, resistin, adiponectin, insulin-like growth factor 1, tissue activator of plasminogen I inhibitor, fibrinogen [4, 28].

At visceral obesity, the main target organs are heart and blood vessels. In heart fat is deposited both inside the cardiomyocytes, leading to fatty myocardial dystrophy, and externally, increasing the thickness of the epicardial fat (EF). Epicardial fat itself develops from brown adipose tissue during embryogenesis and is metabolically more active than subcutaneous adipose tissue. Normally, the EF protects the myocardium and coronary arteries from fatty acids [3]. Since epicardial AT plays an active role in the metabolism of the myocardium, an increase in its volume and activity leads to disruption of the mitochondria. Mitochondrial dysfunction and metabolic disturbance of cardiomyocytes promote systolic cardiac dysfunction in obese patients. At epicardial obesity of heart fibrosis of the myocardium can quickly develop, which accelerates apoptosis of cardiomyocytes. The excess amount of angiotensin II, secreted by the EF, increases the synthesis of fibrosis, which subsequently gives rise to the development of atrial fibrosis and the diastolic function of heart due to the LV myocardial hypertrophy. J. Shirani et al. revealed that excessive fat deposition in the atrial septum increases the prevalence of atrial fibrillation [4, 25-27]. As a result of a pathological increase in the volume of the EF around heart and inside the myocardium, conditions are created for significantly loading the course of IHD, characterized by a more severe lesion of the coronary arteries, more pronounced metabolic disorders, hypercoagulable activity, and shifts in the system of lipid transport of atherogenic orientation [3,4,22].

Currently, epicardial fat is considered as a marker of cardiovascular diseases. A statistically significant relationship with the waist circumference was revealed, with a level of low density lipoproteins. The thicker the epicardial fat, the more pronounced arterial hypertension in patients is and a higher level of insulin. According to a Park study conducted in 2008, for 30 days, in patients with acute coronary syndrome often developed fatal and nonfatal infarcts, cerebral stroke, if the thickness of their epicardial fat was 7.5 mm (against the control group of 5 Mm). According to some data, at the thickness of epicardial fat in 7 mm signs of subclinical atherosclerosis are revealed. According to other data, at epicardial fat in 9 mm with a high probability insulin resistance is diagnosed. In patients with metabolic syndrome, the epicardial fat layer is larger (6 mm) than in patients without metabolic syndrome (4.9 mm). At the moment no clear quantitative value of the layer of epicardial fat has been found [4, 7].

The most significant factors initiating the atherosclerotic process at obesity include: excess of visceral and epicardial fat, resistance index, low level of adiponectin, increased expression of proinflammatory adipokines and cytokines, hypercoagulation, hypertension, hyperglycemia, atrial septal defect [4,7,15- 17].

In the Russian Federation, the prevalence of cardiovascular disease is very high. Ischemic heart disease and cerebrovascular diseases are the main causes of death. In Yakutia over the past 15 years, mortality among the working-age population from diseases of the circulatory system has increased in 2.2 times (in the Russian Federation - 1.7 times).

In order to identify the causes of the development of cardiovascular diseases in Yakutia, a number of studies have been carried out. Medico-social studies have shown that the most significant socio-hygienic factors affecting the formation of circulatory diseases in people of working age are: employment of the population, material wellness, living conditions, the presence of bad habits and the quality of nutrition [10]. Recent populational studies have shown that among men of indigenous nationalities, coronary artery atherosclerosis is less pronounced than in non-indigenous population, with a high prevalence of hypertension (74.7% of cases) and associated hypertrophy of the left departments of heart. There are suggestions that in the development of this phenomenon in the native inhabitants of Yakutia, the presence of insulin resistance, as an independent factor of cardiovascular diseases, probably played a role [13]. Epidemiological studies of the prevalence of the metabolic syndrome and its clinical variants among the indigenous population were also conducted. The most frequent variant of the metabolic syndrome was a combination of abdominal obesity, high blood pressure and lipid metabolism disorders. This combination was more common in young people aged 20-39 years and mainly in women. After 40 years, disorders of carbohydrate metabolism were detected in 52% of the indigenous population [14]. Violations of lipid metabolism are an important issue for residents of Yakutia. In the North, lipids supply the body with the energy that means the transition of the basic metabolism from carbohydrate to fat. In indigenous people, the protein-lipid type of metabolism is genetically determined and is formed in the process of ontogenesis under the influence of external factors. According to the research, in the indigenous and non-indigenous population of Yakutia, an increase in the prevalence of dyslipidemia and differences in the lipid spectrum were detected. As a result of the violation of the ratios of atherogenic and antiatherogenic fractions of lipids, the coefficient of atherogenicity exceeded the permissible norm and was higher for the Yakuts (in the Yakuts in 2.16, for the Russians - 1.9 times) [21].

Cardiovascular diseases are largely a pediatric problem. Considering that overweight in childhood and adolescence is often transformed into obesity in adults, this phenomenon can be considered the metabolic foundation of cardiovascular disease [5, 12, 15-16].

In recent years, convincing evidence has been obtained that the atherosclerotic process leading to CVD

begins in childhood and adolescence and develops over the course of life under the influence of genetic and modifiable risk factors. According to the data of large-scale prospective epidemiological studies, the concept of risk factors, which is the basis for the prevention of CVD, has developed [5, 17, 18, 23]. Later, the concept of risk factors was extrapolated to the child population. As a result of the study, risk factors that retained stability during life were identified. Stable risk factors are hypertension, overweight, hypercholesterolemia, carbohydrate metabolism disorder, low physical activity and smoking among the child population [5, 16]. Despite a variety of risk factors, obesity is the most common risk factor for CVD in children. Critical periods for the debut of obesity are the first year of life, the age of 5-6 years and the period of puberty [8, 9]. The prevalence of excess BM and obesity among boys begins to increase rapidly at the end of puberty (after 15 years). The greater the BMI value in adolescence, the higher it's resistance in subsequent life periods and the higher the risk of excess BM and obesity in young adulthood [8, 9, 15, 16]. The development of metabolic syndrome against obesity in children increases the risk of type 2 diabetes and cardiovascular diseases in young adults. The most frequent complications of obesity (type 2 diabetes, non-alcoholic fatty liver disease, arterial hypertension) can be diagnosed already at the younger school age. Today, the prevalence of childhood obesity has increased significantly in countries with both high and low and middle income countries, and currently solving the problem of childhood obesity is an urgent public health challenge [1, 2, 5].

According to 2010 data, the prevalence of obesity in the Republic Sakha (Yakutia) as a whole was 9.4 among children 0-14 years old, among adolescents - 14.3 per 1000 children of the corresponding age. The statistically significant differences in the average incidence rates for the analyzed period in 3 groups of districts ($p < 0.001$) were revealed. The highest incidence of obesity among children aged 0-14 years was observed in the Arctic group of regions (13.3 per 1000 children), the lowest in the agricultural group (5 per 1000 children). Adolescents had high rates of obesity in the industrial group of districts (17.2 per 1,000 adolescents), the lowest in the agricultural group (11.3 per 1000 adolescents). For 2000-2010 in both age groups, a statistically significant increase in the incidence of obesity was observed in all groups of districts ($p < 0.001$). For example, among children aged 0-14 years, the incidence of obesity in the Arctic group of regions increased three-fold (from 4.6 to 13.3 per 1000), in industrial and agricultural two-fold: from 2.6 to 5 and from 5, 9 to 11.2 per 1.000 children, respectively. Among adolescents, there is also an increase in the incidence of obesity in the dynamics: in the industrial group from 5.9 to 17.2, in the Arctic group - from 4.6 to 14.6 (3 times), in the agricultural group of districts - from 2.6 to 11.3 per 1.000 teenagers (4 times). In the dynamics for 2000-2010 among the children of the Republic Sakha (Yakutia), there is a statistically significant increase in the incidence of obesity. The prevalence of obesity in agricultural areas was statistically significantly lower than in the arctic and industrial groups of areas. These differences may be due to the ethnic composition of the population, the nature of nutrition and the level of motor activity of children in different groups of areas [11].

CONCLUSION

Thus, at obesity a violation of the functional activity of adipose tissue due to excessive accumulation in vital organs leads to their functional and organic lesions, presenting a health risk. It is especially important to note the transformation of childhood obesity into adulthood and the increase in the prevalence of obesity among children. Long-term accumulation of adipose tissue leads to the most persistent damage to human organs and systems, primarily cardiovascular. An important factor is the living conditions. As the researches show, in the residents of the indigenous population of Yakutia specific clinical manifestations of metabolic risk factors for the development of cardiovascular diseases were identified, which may be the reason for its steady growth among the residents of Yakutia.

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