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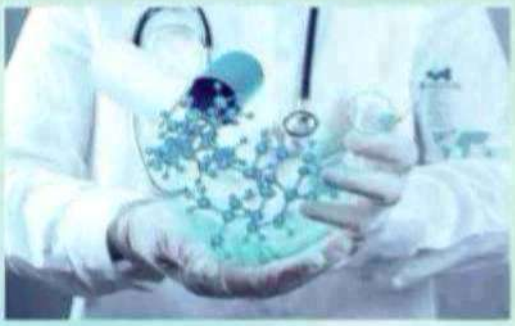

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НОВЫЕ МЕХАНИЗМЫ ФОРМИРОВАНИЯ МЕТАБОЛИЧЕСКОГО СИНДРОМА У ПОДРОСТКОВ

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✓ ***Резюме***

Распространенность МС среди подростков в мире составляет 3–5%, достигая 30% и более у страдающих ожирением. Патогенез МС обусловлен ожирением и хроническим воспалением: избыток висцерального жира приводит к липотоксичности, гиперинсулинемии, нарушению обмена глюкозы и жиров. Инсулинорезистентность и воспалительные цитокины вызывают каскад изменений – от гипергликемии и атерогенной дислипидемии до артериальной гипертензии. Ранняя диагностика и профилактика МС основываются на модификации образа жизни: нормализация питания, повышение физической активности, снижение массы тела. Эти меры позволяют предотвратить долгосрочные осложнения метаболического синдрома.

Ключевые слова: метаболический синдром, подростки, патогенез, инсулинорезистентность

ЎСМИРЛАРДА МЕТАБОЛИК СИНДРОМ ШАКЛЛАНИШИНИНГ ЯНГИ МЕХАНИЗМЛАРИ

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✓ ***Резюме***

Дунёда ўсмирлар орасида МС тарқалиши тахминан 3–5% ни ташкил этади, семиз ўсмирларда эса бу кўрсаткич 30% дан ошиши мумкин. Метаболик синдром патогенези асосида висцерал семизлик ва доимий яллиғланиш ётади: ортиқча ёғ тўқимаси инсулинга нисбатан резистентликка, гиперинсулинемияга, глюкоза ва ёғ алмашинувининг бузилишига олиб келади. Қизларда поликистик тухумдон синдроми ҳам МС хавфини оширади. Метаболик синдромнинг олдини олиш ва даволаш ёшларда асосан турмуш тарзини тузатишга қаратилган: тўғри овқатланиш, жисмоний фаолликни ошириш ва ортиқча вазнни камайтириш. Бу чоралар ўсмирлар орасида метаболик бузилишларни яхшилаб, келажакдаги асоратларнинг олдини олишга хизмат қилади.

Калит сўзлар: метаболик синдром, ўсмирлар, патогенез, инсулин резистентлиги

NEW MECHANISMS OF FORMATION OF METABOLIC SYNDROME IN ADOLESCENTS

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✓ ***Resume***

The prevalence of MetS among youth is about 3–5% globally, but rises to 30% or more in obese adolescents. The pathophysiology of pediatric MetS centers on excess visceral fat and chronic low-grade inflammation leading to insulin resistance. Adipose-derived fatty acids and inflammatory cytokines impair insulin signaling, causing hyperinsulinemia and glucose intolerance, while also promoting atherogenic dyslipidemia (elevated triglycerides, low HDL) and elevated blood pressure. Key risk factors for MetS in adolescence include obesity – especially abdominal obesity – along with physical inactivity and poor diet. Genetic predisposition and family history of cardiometabolic diseases increase MetS susceptibility. Early identification of at-risk adolescents and multidisciplinary preventive strategies are crucial to mitigate the long-term health impact of metabolic syndrome in the young population.

Keywords: metabolic syndrome, adolescents, pathogenesis, insulin resistance

Relevance

In children and adolescents, MS is also considered as a factor of early increase in cardiometabolic risk [3]. However, the use of the concept of MS in pediatrics is controversial – there is no single diagnostic criterion for young patients [4]. More than 40 different definitions of childhood MS have been proposed [5], and there is still no consensus on the age and thresholds for diagnosing MS in the youth population. For example, the International Diabetes Federation (IDF) in 2007 She recommended diagnosing MS only in children over 10 years of age (with three or more abnormalities with mandatory central obesity) and not using the diagnosis at an earlier age [6]. Nevertheless, already in adolescence, the combination of obesity with hypertension, impaired glucose tolerance and dyslipidemia is quite common. Moreover, it has been shown that in children with MS, these disorders often persist into adulthood: for example, in longitudinal studies, the presence of MS in childhood doubled the risk of developing diabetes and cardiovascular complications after 25-30 years [7]. Due to the epidemic of obesity among young people, interest in MS in pediatrics has increased dramatically [8], since early prevention and correction of MS components can reduce the future incidence of CVD [9]. This article examines the prevalence, pathogenesis, and risk factors of MS in adolescents based on current data.

The purpose of the study: to study the pathogenetic features of metabolic syndrome in adolescents.

Research materials and methods: A review of foreign materials and literature sources on medical research from the MEDLINE and PubMed databases over the past 5 years (2021-2025) has been conducted.

Result and discussions

The prevalence of MS in adolescents varies significantly depending on the criteria used and the characteristics of the population [10,11]. Globally, the proportion of adolescents with MS is estimated at about 5% (as of 2020) [12]. MS is less common in children under 12 years of age (~3%), while it is more common in people aged 13-18 (~5%) [12]. According to a 2021 meta-analysis, the prevalence of MS in the adolescent population ranges from 0.3% to 26% in different studies [13]. This gap is caused by differences in diagnostic definitions: for example, according to the criteria of the IDF 2007. The incidence of MS in adolescents was 0.3–9.5%, and when using milder de Ferranti criteria, it ranged from 4% to 26% [11]. In most samples, about 3-5% of all adolescents have MS [14].

Among overweight and obese children, the rates are significantly higher: for example, the median prevalence of MS is ~12% in overweight and up to 29-30% in obese adolescents [14]. Close to 90% of obese adolescents have at least one component of MS [15], and about a third or half have the entire syndrome. A clear relationship between the degree of obesity and the frequency of MS has been confirmed [16]. In addition, there are gender, age and ethnic differences: in general, boys are slightly more susceptible to MS than girls, especially during puberty [17]. Among ethnic groups, representatives of Latin American, Asian, and some other populations have a high risk [18].

This is attributed to a combination of genetic characteristics of fat distribution and a diet rich in sugar and fats [19]. In the United States, the prevalence of MS among adolescents is estimated at 4-5% and generally remained stable from 1999 to 2018[20]. In European countries, the figures are close to

5-6% [11]. In Asian and Middle Eastern countries, MS is especially common in obese adolescents, up to 30-40% or more [21]. It should also be noted that some of the conditions associated with MS appear only with age. For example, glucose tolerance may decrease gradually and manifest as diabetes in adulthood, despite normoglycemia in adolescents with MS [22][23]. Nevertheless, even in youth, the combination of MS components poses a serious health threat – according to the American Heart Association, adolescent MS is associated with a higher risk of early atherosclerosis and mortality at a young age [8]. MS in female adolescents with polycystic ovaries (PCOS) deserves special mention. Girls with PCOS have metabolic syndrome 3-4 times more often than their peers without this syndrome [24]. According to the results of a meta-analysis in 2023, about 30-40% of adolescents suffering from PCOS have concomitant MS, especially in the presence of obesity [25]. These data emphasize the need for early detection of MS components in girls with endocrine disorders (hyperandrogenism). to avoid serious consequences.

MS is considered as a clinical manifestation of insulin resistance (IR) against the background of excess visceral fat and chronic inflammation [26][27]. The pathogenesis is based on obesity, especially abdominal obesity, which triggers a cascade of metabolic disorders. With an overabundance of nutrition and low physical activity, excess triglycerides accumulate in the body, which are deposited not only in adipose tissue, but also ectopically in muscles, liver, and pancreas [28]. An excess of free fatty acids and lipids leads to “lipotoxicity” – damage to beta cells of the pancreas and a decrease in their ability to secrete insulin, as well as disruption of the transmission of insulin signals in muscle and liver tissue [28]. Insulin resistance develops: cells respond less well to insulin, and the pancreas is forced to produce more of the hormone (hyperinsulinemia) [29].

IR in the liver leads to insufficient suppression of gluconeogenesis, which explains the tendency to fasting hyperglycemia in patients with MS [30]. At the same time, excess insulin activates lipogenesis genes in the liver, which increases the synthesis of triglycerides and the secretion of very low–density lipoproteins (VLDL) [11]. Atherogenic dyslipidemia occurs: hypertriglyceridemia and a decrease in HDL cholesterol [11][27]. Increased secretion of VLDL triglyceride particles leads to the formation of small dense LDL particles, the most atherogenic fractions of cholesterol l [12]. At the same time, hyperinsulinemia contributes to arterial hypertension by activating the sympathetic nervous system, increasing sodium reabsorption in the kidneys and hypertrophy of vascular smooth muscle cells [13]. Normally, insulin has a vasodilating effect by stimulating the release of nitric oxide in the endothelium; with IR, this function is disrupted and endothelial dysfunction develops [14]. Thus, the vicious circle of IR leads to the simultaneous development of the main components of MS: glucose intolerance (hyperglycemia), dyslipidemia, and elevated blood pressure [23,25]. Visceral adipose tissue, an active endocrine organ, plays an essential role in pathogenesis. With obesity, adipocytes increase in size, they decrease the production of protective adipokine adiponectin and increase the synthesis of proinflammatory cytokines (TNF-α, interleukin-6, etc.) [27]. This leads to a chronic state of low-grade systemic inflammation, which involves macrophages and other immune cells in adipose tissue and exacerbates insulin resistance [27]. A decrease in the level of adiponectin additionally increases inflammation and IR [16]. Visceral obesity is also associated with activation of the renin-angiotensin-aldosterone system (RAAS): adipose tissue expresses angiotensinogen, which is converted into angiotensin II, which causes vasoconstriction, sodium retention, and increased blood pressure [17,18].

Thus, obesity and IR initiate a cascade of metabolic and vascular changes that are clinically manifested by MS and increase the risk of diabetes and atherosclerosis [39][38]. Recent studies on the pathogenesis of MS also note the role of oxidative stress and imbalance of the intestinal microbiota in exacerbating IR [20,21]. In addition, it has been shown that the tendency to MS is laid very early – even before birth. Adverse effects in the prenatal period, such as obesity or diabetes mellitus in the mother during pregnancy, increase the risk of metabolic disorders in the offspring [22,23]. Children born to mothers with gestational diabetes have increased fat deposition and a higher incidence of obesity and MS at puberty [14]. It is assumed that such effects are associated with epigenetic restructuring: an excess of nutrients in the mother causes changes in DNA methylation and gene regulation in the fetus, “programming” his body to develop obesity and IR in the future [15,16]. Thus, it was found that children born to obese mothers have a larger adipocyte size and pronounced IR already in early childhood [17]. Thus, the pathogenesis of MS in adolescents is a multifaceted process

that includes genetic predisposition, environmental and lifestyle influences, hormonal changes during puberty, and long–term effects of the perinatal period.

A key risk factor for MS at any age is overweight, especially abdominal obesity [18,19]. The probability of developing MS is directly proportional to the degree of obesity: with a body mass index (BMI) >95th percentile (obesity), a child's chance of developing MS increases many times compared with peers with normal weight [18]. In addition to body weight, fat localization is important: the accumulation of visceral adipose tissue increases cardiometabolic risks even in not overweight children [50]. To assess central obesity in pediatrics, waist circumference (OT) and waist/height ratio are used. If this index is ≥0.5, the risk of MS is significantly increased [21].

Heredity also plays a role: children with family problems (early cardiovascular diseases, type 2 diabetes mellitus, and parental obesity) are more prone to metabolic disorders. For example, it has been shown that the presence of obesity in parents increases the risk of MS in a child by 2-3 times [22]. The intrauterine factors mentioned above (diabetes and excess weight of a pregnant woman, low or overweight fetus at birth) also affect the future metabolism of children [23,24]. Genetic predisposition is manifested through polymorphisms of genes that affect appetite, adipokine profile, etc. However, modifiable environmental factors are much more significant [23].

The latter include, first of all, the peculiarities of a teenager's diet and physical activity. Poor nutrition – excess calories, frequent consumption of sugary sodas, fast food, foods high in simple carbohydrates and saturated fats – is directly associated with the development of obesity, hypertension and dyslipidemia [15,16]. Thus, a large study has shown that the "Western" type of diet (lots of red meat, sugar and refined grains, lack of vegetables and fiber) is associated with a higher risk of MS in adolescent s [17,18]. On the contrary, a balanced diet like the Mediterranean diet (vegetables, fruits, whole grains, fish, nuts, vegetable oils) It helps to reduce the level of inflammation and improves metabolic parameters [19,20]. The second most important factor is low physical activity. A sedentary lifestyle and a lack of regular exercise lead to chronic energy imbalance and weight gain, which in children is closely associated with an increase in the prevalence of MS [11].

Observations show that adolescents who lead a sedentary lifestyle are much more likely to experience obesity and insulin resistance than their more active peers [62]. Insufficient sleep duration is also recognized as a risk factor for obesity and metabolic disorders in young people [63]. Hormonal changes during puberty can temporarily reduce insulin sensitivity (especially in girls), which, in the presence of obesity, exacerbates the metabolic imbalance at puberty. In girls, polycystic ovary syndrome (PCOS) is accompanied by an increase in androgen and IR levels, regardless of body weight, therefore PCOS is considered an independent risk factor for MS [14]. As noted, the incidence of MS in adolescents with PCOS is three times higher than that in healthy girls [24], and the presence of obesity further increases this risk. In this regard, endocrinologists recommend regularly checking the indicators of lipid metabolism and blood pressure in all girls with PCOS for early detection of MS [15]. Finally, chronic stress and psychological aspects are an important risk factor. It has been noted that adolescents with depression and eating disorders are more likely to have obesity and metabolic disorders. Thus, the risk group for MS includes adolescents with obesity (especially abdominal), with burdened heredity, reduced physical activity and unhealthy diet, as well as girls with hyperandrogenic conditions. Identifying such adolescents and targeting their lifestyle changes is the most important task of preventing MS.

Conclusion

Metabolic syndrome in adolescents is a serious medical and social problem that arises against the background of the epidemic of childhood obesity. Although clear diagnostic criteria for childhood have not yet been developed, the concept of MS is useful for early detection of a combination of risk factors leading to the premature development of diabetes mellitus and atherosclerosis [24]. Numerous studies confirm that the clinical "coexistence" of obesity, hypertension, dyslipidemia and hyperinsulinemia in adolescence is directly related to a worse prognosis in adulthood [7,8]. In this regard, the prevention and correction of individual components of MS in children is a priority strategy. The main measures are considered to be non–drug interventions - lifestyle changes, including a healthy balanced diet and sufficient physical activity [15,19]. A decrease in body weight of even 5-10% leads to a significant improvement in the metabolic profile of obese adolescents [11,17]. The

results of a number of interventions indicate that a decrease in visceral fat is accompanied by normalization of insulin sensitivity, a decrease in triglycerides and blood pressure, that is, it actually eliminates the manifestations of MS [18,19]. Therefore, all international guidelines recommend, first of all, weight correction and increased activity in children with signs of MS [10]. Drug therapy (metformin to improve IR, hypolipidemic or antihypertensive drugs) in adolescence is used only to a limited extent and strictly according to indications, as part of the treatment of certain diagnoses (for example, hypertension) [10]. In cases of severe obesity in adolescents, bariatric treatment may be discussed if conservative measures are ineffective [11]. In general, emphasis should be placed on early detection of overweight children with signs of metabolic disorders and on comprehensive preventive work – dietary counseling for families, the development of physical education programs, and training in healthy lifestyle skills. The success of such interventions largely depends on an interdisciplinary approach: pediatricians, endocrinologists, cardiologists, nutritionists and psychologists should jointly supervise adolescents at risk, involving their parents in the process [12,13]. The modern view of pediatric MS is that it can be prevented by forming a culture of healthy eating and activity in children from a young age. Solving this problem will reduce the burden of diabetes and cardiovascular diseases in future generations.

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