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ЖУРНАЛ ГЕПАТО-ГАСТРОЭНТЕРОЛОГИЧЕСКИХ ИССЛЕДОВАНИЙ

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OBESITY AND VITAMIN D DEFICIENCY IN CHILDREN AND ADOLESCENTS, THE PRESENT CONDITION OF THE PROBLEMS

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ANNOTATION

This literature review examines the significance of vitamin D deficiency in children, its role in the development of obesity, and other somatic pathologies. It provides information on the prevalence of vitamin D deficiency in children and adolescents, risk factors, and the pathogenesis of vitamin D deficiency in children with obesity. Also discussed are methods for correcting vitamin D deficiency in both obesity and other metabolic disorders.

Keywords: obesity in children, vitamin D, treatment of vitamin D deficiency.
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BOLALAR VA O‘SMIRLARDA SEMIZLIK VA D VITAMINI YETISHMOVCHILIGI: MUAMMONING HOZIRGI HOLATI

ANNOTATSIYA

Ушбу адабиётлар шарҳида, болаларда витамин Д етишмовчилиги, унинг семизлик ва бошқа соматик патологиялар ривожланишида тутган ўрни ҳақида ёритилган. Болалар ва ўсмирларда витамин Д дефицити тарқалиш даражаси ҳақида маълумотлар, семиз болаларда витамин Д етишмовчилиги ривожланишининг хавф омиллари ва патогенези. Шу билан биргаликда витамин Д коррекциясининг, семизликда ва бошқа моддалар алмашинуви бузилишида, ўтказилиш усуллари кўрсатилган.

Калит сўзлар: Болаларда семизлик, витамин Д, витамина Д дефицитининг коррекцияси.

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ОЖИРЕНИЕ И ДЕФИЦИТ ВИТАМИНА D У ДЕТЕЙ И ПОДРОСТКОВ: СОВРЕМЕННОЕ СОСТОЯНИЕ ПРОБЛЕМЫ

АННОТАЦИЯ

Данный обзор литературы, посвящён актуальности проблемы дефицита витамина Д у детей, его роли в развитии ожирения и другой соматической патологии. Приведены сведения о распространённости дефицита витамина Д, у детей и подростков, факторы риска и патогенез развития дефицита витамина Д при ожирении у детей. Также обсуждаются методы коррекции дефицита витамина D как при ожирении, так и других нарушений обмена веществ.

Ключевые слова: ожирение у детей, витамин Д, коррекция дефицита витамина Д.

According to the World Health Organization (WHO), the prevalence of overweight and obesity among children and adolescents remains high. In 2016, 41 million children under the age of five and 340 million children aged 5–19 were diagnosed with these conditions worldwide. This underscores obesity as a major global public health concern in children.

In recent years, complications traditionally associated with adulthood—such as arterial hypertension, type 2 diabetes mellitus, non-alcoholic fatty liver disease, and cholelithiasis—have been increasingly observed in obese children, particularly adolescents. Additionally, population-based studies across diverse ethnic and age groups reveal a strong correlation between obesity and inadequate vitamin D levels [9]. Currently, obesity is recognized as a major risk factor for vitamin D deficiency and secondary hyperparathyroidism. Individuals with morbid obesity are particularly vulnerable to severe vitamin D deficiency. Beyond its role in bone health, vitamin D is also implicated in the pathogenesis of various multifactorial diseases. Notably, vitamin D influences blood lipid composition, blood pressure [23], cognitive function [1], and cancer risk [12]. Furthermore, vitamin D deficiency

has been linked to an increased susceptibility to infectious diseases, including tuberculosis [11].

Vitamin D deficiency is prevalent worldwide, including in Western European countries. For instance, a recent study involving a cohort of 1,006 adolescents aged 12–17 years from nine European Union (EU) countries reported widespread vitamin D insufficiency. Vitamin D levels were classified as optimal (>30 ng/ml), insufficient (20–30 ng/ml), deficient (10–20 ng/ml), and severely deficient (<10 ng/ml). The average serum 25-(OH)-D level in the cohort was 22.8 ng/ml, with 80% of participants exhibiting suboptimal levels (39% insufficient, 27% deficient, and 15% severely deficient) [3]. These findings underscore the alarming prevalence of vitamin D insufficiency among adolescents, even in economically developed EU countries.

Recent population-based studies also indicate a high prevalence of hypovitaminosis D among children in other regions. In the United States, 61% of children have 25-(OH)-D levels between 15–29 ng/ml, while 9% have levels below 15 ng/ml [14]. Similarly, an epidemiological study in Brazil—despite the country's high solar exposure—revealed that 14% of children under 10 years old and 24% of adolescents had vitamin D levels below 20 ng/ml [15]. In the United Arab Emirates, an assessment of vitamin D status in four pediatric age groups found that children aged 8–14 years had a higher prevalence of deficiency compared to younger children (2–7 years). Thus, adolescents are at the highest risk of vitamin D deficiency, a concern further exacerbated by the fact that their increased vitamin D requirements are often overlooked [41].

Vitamin D is classified as a secosteroid. The two most studied forms in humans are vitamin D2 (ergocalciferol), obtained from dietary sources, and vitamin D3 (cholecalciferol), synthesized in the skin from 7-dehydrocholesterol upon exposure to ultraviolet B (UVB) radiation. Vitamin D is sequestered in adipose tissue and transported in the bloodstream via vitamin D-binding protein [20].

Vitamin D3 is biologically inactive and requires two hydroxylation steps to become active [30]. The first occurs in the liver, where cholecalciferol is converted to calcidiol (25(OH)D3) by 25-hydroxylase. The second takes place in the kidneys, where calcidiol is converted into the biologically active form, calcitriol (1,25(OH)2D3), by 1α-hydroxylase. Calcitriol binds to its receptor, forms a heterodimeric complex with the retinoid X receptor, and interacts with vitamin D response elements on DNA, thereby regulating gene transcription, including that of the Kl (Clotho) gene [20, 30].

Vitamin D receptors (VDRs) are widely distributed across various organs, including the cardiovascular system, where they are expressed in vascular smooth muscle cells [18, 34], endothelial cells [19], and cardiomyocytes [40]. Experimental studies have demonstrated that, in vitro, the active form of vitamin D influences cardiomyocyte and vascular smooth muscle cell activity, modulates cell proliferation [27, 28], suppresses the renin-angiotensin system [17], regulates calcium absorption in vascular smooth muscle cells [6], inhibits cytokine release from lymphocytes [33], and affects inflammation and lipid metabolism [29].

Serum 25(OH)D3 levels, the primary circulating form of vitamin D, reflect both cutaneous synthesis and dietary intake [20]. According to the WHO, more than one billion people worldwide suffer from vitamin D deficiency, highlighting the need for increased awareness and preventive measures to address this global health issue.

The risk factors contributing to vitamin D deficiency in the body include:

- Geographic location of residence
- Season of the year
- Skin pigmentation intensity
- Maternal vitamin D status
- Consumption of vitamin D-fortified foods
- Overall nutritional status
- Presence of comorbid conditions
- Genetic polymorphisms affecting vitamin D receptors

Several factors contributing to vitamin D deficiency in children and adolescents should be considered[22]:

1. **Reduced Vitamin D Intake or Synthesis:**
   - Birth to a mother with vitamin D deficiency
   - Prolonged exclusive breastfeeding
   - Dark skin pigmentation
   - Limited sun exposure due to excessive sunscreen use, protective clothing, chronic illnesses, or frequent hospitalizations
   - Inadequate intake of vitamin D-rich foods
2. **Intestinal Dysfunction and Malabsorption:**
   - Celiac disease, food allergies and exudative enteropathy
   - Exocrine pancreatic insufficiency (e.g., cystic fibrosis, Schwachman-Diamond syndrome)
   - Biliary obstruction
3. **Decreased Synthesis or Increased Degradation of 25-(OH)-D or 1,25-(OH)2D:**
   - Chronic liver and kidney diseases
   - Medications that accelerate vitamin D metabolism (e.g., rifampicin, isoniazid, anticonvulsants)

Obesity and Vitamin D Deficiency: Obesity-related vitamin D deficiency is driven by multiple physiological mechanisms. Vitamin D, being fat-soluble, is sequestered in the larger volume of adipose tissue in obese individuals, leading to reduced plasma concentrations. Additionally, obese individuals often wear more concealing clothing and spend less time outdoors, limiting natural vitamin D synthesis through sun exposure.

A study of 149 children aged 8–13 years demonstrated an inverse correlation between vitamin D levels and triglyceride levels (correlation coefficient r = -0.86, p = 0.01). This effect remained statistically significant after adjusting for age, gender, BMI, and physical activity [32].

To further investigate the mechanisms underlying vitamin D deficiency in obesity, Worthman J. et al. examined serum concentrations of D2, D3, and 25-(OH)-D in obese and normal-weight individuals following ultraviolet (UV) exposure and oral ergocalciferol supplementation [36]. The study included 19 healthy participants (BMI ≤25) and 19 obese participants (BMI >30), all Caucasian with skin types II and III. Serum vitamin D3 concentrations typically peak 24 hours post-UV exposure. Blood samples were collected one hour before and one day after exposure to measure vitamin D synthesis and transport from the skin to the bloodstream. Basal vitamin D3 concentrations did not significantly differ between obese and control groups. However, the increase in vitamin D3 levels post-UV exposure was significantly lower in obese individuals than in non-obese individuals (6.7±1.4 ng/ml vs. 15.3±2.1 ng/ml, p=0.0029). Although obese individuals have a greater body surface area, their post-UV exposure vitamin D3 increase was 57% lower than that of non-obese individuals, suggesting impaired bioavailability rather than synthesis capacity. [24-25].

Earlier research from the 1980s and 1990s indicated that obesity does not affect the content of 7-dehydrocholesterol (the precursor of vitamin D3) in the skin, nor does it impair vitamin D synthesis. This suggests that the primary issue in obesity is impaired transport of vitamin D from the skin to the bloodstream. It is hypothesized that subcutaneous fat absorbs a greater proportion of the vitamin produced in the skin, thereby limiting its bioavailability.

Findings by Arunabh S. et al. further support the hypothesis that reduced serum 25-(OH)-D levels in obesity result from tissue redistribution within a larger volume of adipose tissue [2]. Their study examined the relationship between 25-(OH)-D levels and body fat percentage in 410 healthy women (BMI range: 17–30), revealing an inverse correlation between adipose tissue percentage and 25-(OH)-D levels.

A large-scale study confirmed that serum 25-(OH)-D levels decline as BMI increases, while HbA1c levels tend to rise. Among obese individuals, 80% had serum 25-(OH)-D levels below 75 nmol/L, compared to 68% of non-obese individuals ($p < 0.0001$). Additionally, patients with type 2 diabetes or HbA1c levels exceeding 7% exhibited significantly lower 25-(OH)-D levels than those with normal glucose metabolism (36.9 nmol/L vs. 52 nmol/L).

To further evaluate vitamin D metabolism**,** researchers analyzed 2,126 patients at the Oslo Clinic for Metabolism and Medical Lifestyle

Management, examining seasonal fluctuations in 25-(OH)-D3 levels relative to BMI, gender, and age [16]. Multiple regression analysis revealed that for every 1 kg/m² increase in BMI, serum vitamin D3 concentration decreased by 0.74 nmol/L. Similarly, each 1 cm increase in waist circumference, serum vitamin D3 levels declined by 0.29 nmol/L. These findings highlight the association between obesity (measured by BMI and waist circumference) and vitamin D3 deficiency.

Despite higher total food intake, obese individuals frequently exhibit deficiencies in essential nutrients, including vitamin D. Meta-analyses of randomized, placebo-controlled trials suggest that a daily intake of at least 700–800 IU of vitamin D is required to prevent bone mineral density loss and reduce the risk of falls and fractures. Since vitamin D enhances calcium absorption, its effectiveness is optimized when combined with at least 1,000 mg of calcium per day [13]. Ongoing debate exists regarding the optimal daily dosage of vitamin D. Current recommendations for correcting vitamin D deficiency, unfortunately, lack strong clinical evidence. In cases of confirmed deficiency, ergocalciferol is typically prescribed at 50,000 IU per week for eight weeks. Once 25(OH)D levels normalize, maintenance therapy with 800–1,000 IU/day of cholecalciferol is recommended [4]. According to Cannell J., individuals with established vitamin D deficiency may require higher doses, ranging from 2,000 to 7,000 IU per day, to maintain 25(OH)D levels within 40–70 ng/ml (100–175 nmol/L) [7,8]. As previously noted, obese individuals have an increased need for vitamin D, which should be considered when prescribing preventive or therapeutic supplementation. The target 25(OH)D level should not fall below 30 ng/ml (75 nmol/L), and achieving this may require doses exceeding 800–1,000 IU per day [5]. Some clinical studies suggest that 2,000–4,000 IU/day may be effective in managing obesity and diabetes. A 12-week study involving obese adolescents receiving 2,000 IU/day of vitamin D3 or a placebo demonstrated a significant serum 25(OH)D increase of +6 ng/ml ($p < 0.001$) [26]. Another 12-month study showed that a daily intake of 3,000 IU of vitamin D3 increased adiponectin levels ($p < 0.02$) in vitamin D-deficient obese children (<15 ng/ml) [35]. Additionally, supplementation with 25,000 IU/week (~4,000 IU/day) for nine weeks in a group of 109 obese children (aged 8–18 years) with vitamin D deficiency (<50 nmol/L) reduced deficiency cases to 25% without adverse effects [31]. A study assessing daily vitamin D intake in pregnant women found that a dose of 2,000 IU/day was the most effective in preventing deficiency. Among 91 participants, the incidence of vitamin D deficiency (<20 ng/ml) was 20% in the 2,000 IU/day group, significantly lower than the 58% and 61% observed in the 600 IU/day and 1,200 IU/day groups, respectively ($p = 0.03$). Similarly, among newborns, vitamin D deficiency was detected in only 9% of those born to mothers receiving 2,000 IU/day, compared to 74% and 48% in the lower-dose groups ($p = 0.006$). These findings suggest that a minimum intake of 2,000 IU/day is necessary to maintain optimal vitamin D levels during pregnancy and early infancy [38].

Another study examined individuals at increased risk for diabetes, defined by glycated hemoglobin levels between 5.8% and 6.9%, who also had vitamin D deficiency (25-OHD <30 ng/ml). Participants were randomized to receive either weekly vitamin D supplementation (88,865 IU/week, n = 56) or a placebo (n = 53) for 12 months. Within three months, mean 25-OHD levels in the supplemented group rose from 22 ng/ml to 70 ng/ml. After 12 months, glycated hemoglobin levels showed a significant reduction (-0.2%) in the vitamin D group, highlighting its potential role in metabolic regulation [10].

It is essential to emphasize that administering single megadoses of vitamin D is both risky and often ineffective. A randomized trial involving 61 individuals with type 2 diabetes mellitus (T2DM) investigated the effects of a single high-dose vitamin D administration (100,000 IU or 200,000 IU) [37]. Beyond the ethical concerns surrounding such large doses, described as a form of "human experimentation" [39], the study [21] found no measurable improvements in endothelial function, insulin resistance, or glycated hemoglobin levels. These findings reinforce the notion that long-term, consistent vitamin D supplementation at physiological doses remains the safest and most effective strategy.

Conclusion: The global prevalence of obesity continues to rise, affecting individuals at increasingly younger ages. The primary contributors to obesity in children and adolescents are modifiable risk factors, including excessive intake of carbohydrates and saturated fats, micronutrient deficiencies, physical inactivity, alcohol consumption, and smoking. Among these, vitamin D deficiency plays a significant role in increasing the risk of diabetes. Vitamin D has widespread effects on the molecular physiology of cells and tissues. Numerous studies support its role in preventing and managing obesity and related complications. Early detection and correction of vitamin D deficiency, particularly in high-risk groups such as children and adolescents, can reduce the likelihood of chronic diseases in adulthood. This, in turn, can lower healthcare costs. Optimizing vitamin D intake through public health policies and regulatory measures is a crucial step toward improving overall health outcomes.

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