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Serum 25-Hydroxyvitamin D Levels in Children Aged 3-17 Years in Southern Region of Ukraine and Their Association with Anthropometric Z-Scores: Establishing a Predictive Threshold

Introduction. Vitamin D (VD) deficiency ranks among the most prevalent nutritional disorders affecting paediatric populations worldwide [5, 7]. According to international reviews, a considerable proportion of children and adolescents present with 25-hydroxyvitamin D (25(OH)D) levels below recommended thresholds. The prevalence of both deficiency and insufficiency rises with age, particularly among adolescents in temperate latitude countries [6, 7]. In regions situated between 40.0 ° and 60.0 ° north latitude - a category that includes Ukraine - high rates of children with 25(OH)D levels below 50.0 nmol/L have been documented [3, 7].

In Ukraine, this issue carries particular weight. Findings from a multicentre cross-sectional study of children and adolescents aged 1-17 years revealed that 23.5 % (95.0 % confidence interval [CI]: 21.0-26.0 %) of those examined had reduced 25(OH)D concentrations below 30.0 nmol/L. The median level in this population was 65.4 nmol/L (interquartile range [IQR]: 49.5-85.6). Severe deficiency was recorded in 2.9 % of children, moderate deficiency in 23.0 %, and insufficiency in a further 37.0 %, collectively reflecting a high burden of suboptimal VD status [10, 12]. The winter and spring months are especially critical: the concept of "winter vitamin D" stems from the fact that at Ukraine's latitudes, the ultraviolet index falls below 3.0 during the cold season, which is insufficient for effective cutaneous synthesis of cholecalciferol from October through March. This results in a seasonal drop in blood 25(OH)D levels to 30.0-40.0 % of summer values [3, 7]. Even in southern Ukraine, despite geographically more favourable insolation conditions, a high prevalence of hypovitaminosis D (25(OH)D <50.0 nmol/L) has been observed - 71.9 % overall, rising to 81.2 % among adolescents aged 15-17 years. In the adult population of the same region, low VD status is associ-

ated with dyslipidaemia, pointing to the systemic nature of the problem [9, 10]. Dietary VD intake in the examined southern Ukraine cohort amounted to only 45.1 ± 30.1 IU/day and showed no correlation with blood 25(OH)D levels ($r = 0.046$, $p = 0.281$). This finding confirms the dominant role of endogenous synthesis and explains children's vulnerability under conditions of limited sun exposure. Current strategies for preventing VD deficiency in children - taking into account both dietary intake and insolation levels - are thoroughly reviewed in the literature, which underscores the limited contribution of nutritional sources alone [8].

Physical development serves as an integral indicator of a child's health, reflecting how somatic growth and body weight align with age- and sex-specific norms. Assessment using standardised World Health Organization (WHO) Z-scores - height-for-age (HFA), weight-for-age (WFA), body mass index (BMI-for-age), and waist-to-height ratio (WtR) - enables objective, quantitative characterisation of physical development deviations across all age subgroups [5]. Among the child population of southern Ukraine, a phenomenon of "double burden of malnutrition" has been described: 21.7 % of children are overweight or obese (BMI > +1.0 standard deviation (SD) according to WHO criteria), while 21.2 % are underweight (BMI < -1.0 SD). These figures considerably exceed the average European rates reported by the Childhood Obesity Surveillance Initiative (COSI) and indicate a deepening nutritional imbalance in the region [3].

Vitamin D exerts its primary effects through the nuclear VD receptor (VDR), which is expressed in more than 36 tissue types and regulates the transcription of over 200 genes [5]. Its active form, $1,25(\text{OH})_2\text{D}$ (calcitriol), stimulates trans-epithelial calcium transport via transient receptor potential vanilloid channels (TRPV5/TRPV6),

enhances Ca^{2+} reabsorption in the renal tubules, and maintains optimal calcium-phosphate homeostasis - essential for bone mineralisation and linear growth [3, 12]. Under conditions of VD deficiency, secondary hyperparathyroidism mobilises calcium from bone, suppresses mineralisation, and directly affects bone mass parameters and linear growth rates [12]. Furthermore, $1.25(\text{OH})_2\text{D}$ influences adipocyte differentiation through VDR-mediated pathways, reducing the activity of peroxisome proliferator-activated receptor gamma (PPAR- γ) and CCAAT/enhancer-binding protein alpha (C/EBP- α). In VD deficiency, this inhibitory effect is weakened, leading to excessive adipose tissue accumulation. Adipose tissue itself, by sequestering the fat-soluble vitamin, further lowers circulating VD levels through a "volumetric dilution" mechanism [1, 4].

The relationship between VD status and physical development indicators has been confirmed in numerous clinical studies. Systematic reviews and meta-analyses show that children and adolescents with obesity have a 41.0 % higher risk of VD deficiency compared to their normal-weight peers, and lower 25(OH)D levels are associated with abdominal obesity [4, 13]. Among Ukrainian adolescents, the proportion of VD deficiency stands at 70.7 % in those who are overweight and 77.1 % in those with obesity, compared with 57.3 % in individuals of normal weight. Overweight and obese children have significantly lower mean 25(OH)D levels than their normal-weight counterparts, and the proportion with reduced levels (<50.0 nmol/L) in these groups exceeds 80.0 %, confirming a close link between VD status and anthropometric measures [8, 9, 11]. Some studies also point to a high prevalence of VD deficiency and insufficiency in children with short stature and growth retardation. An analysis of the association between VD levels and linear growth in children with various forms of short stature found that VD deficiency occurs in approximately 47.0-60.0 % of such patients - significantly higher than in the general paediatric population [2]. A bidirectional causal relationship between obesity and VD status has been demonstrated in studies employing genetic methods: obesity is associated with lower 25(OH)D levels, while the reverse effect is less pronounced but pathogenetically plausible [4].

Despite the substantial body of evidence on associations between VD status and anthropometric parameters, the literature contains few quantitatively defined predictive thresholds for 25(OH)D that could help identify children at risk of abnormal physical development in routine paediatric practice [3, 7]. Receiver operating characteristic (ROC) analysis is a powerful statistical tool for assessing the diagnostic and predictive value of biomarkers and for determining optimal cut-off points [4]. Establishing a predictive threshold for 25(OH)D for specific anthropometric endpoints would enable standardisation of screening and preventive measures for physical development abnormalities and would provide a rationale for target VD levels within regional prevention programmes.

The aim of the study. To determine the relationship between serum 25-hydroxyvitamin D levels in children aged 3-17 years from the Southern region of Ukraine and their physical development parameters, and to establish a prognostic threshold of 25-hydroxyvitamin D concentration for identifying the risk of deviations in anthropometric Z-scores.

Materials and methods. *Study Design and Setting.* A single-centre cross-sectional study was conducted, incorporating both prospective and retrospective elements, in accordance with the Strengthening the Reporting of Observational Studies in Epidemiology (STROBE) guidelines. The study included 562 children aged 3-17 years (289 boys, 51.4 %; 273 girls, 48.6 %), divided into four age groups: 3-6 years ($n = 158$; 28.1 %), 7-10 years ($n = 140$; 24.9 %), 11-14 years ($n = 152$; 27.0 %), and 15-17 years ($n = 112$; 19.9 %). Participant recruitment took place between 2022 and 2025 at paediatric healthcare facilities in the southern region of Ukraine. Sample size was calculated using G*Power software (version 3.1) to detect a correlation of $r = 0.20-0.30$ between serum 25(OH)D levels and anthropometric Z-scores, with $\alpha = 0.05$ and power set at 80.0-85.0 %. The minimum required sample was estimated at 450 children. An additional reference dataset of laboratory 25(OH)D measurements from 2022-2025 ($n \approx 70,000$) was compiled for external validation of the findings.

Participants. Children were eligible for inclusion if they: (1) were in relative good health with no acute illness at the time of examination; (2) had not received VD supplements exceeding 1,000 IU/day for three months before; and (3) had no chronic conditions known to significantly interfere with VD metabolism or physical development (e.g., kidney or liver disease, malabsorption syndrome, hereditary forms of rickets, or connective tissue disorders). Children were excluded if they were taking glucocorticoids, anticonvulsants, or any medications that impair fat absorption. All participants were subsequently categorised into three groups based on their VD status: deficient, insufficient, or sufficient.

Anthropometric Measurements. Height (measured to the nearest 0.1 cm using a vertical stadiometer), body weight (to the nearest 0.1 kg), and waist circumference (to the nearest 0.5 cm) were recorded using standardised methods in the morning, under fasting conditions, at an ambient temperature of 20.0-24.0 °C. Skinfold thickness was also measured following a standardised protocol. Physical development was assessed according to WHO international standards: the WHO Child Growth Standards (2006) for children aged 0-5 years and the WHO Growth Reference (2007) for those aged 5-19 years. Four primary Z-scores were calculated: HFA, WFA, BMI-for-age, and WHtR. Developmental deviations were defined as Z-scores below 2 SD (stunted growth or underweight) or above +2 SD (obesity) relative to the WHO reference population median. For ROC analysis, a more sensitive threshold of $> +1$ SD was also used as a marker of overweight risk. A WHtR value above 0.50 was considered indicative of central obesity.

Laboratory Assessment. Serum 25(OH)D concentrations were measured using electrochemiluminescence immunoassay (ECLIA) with standardised reagents, in compliance with VD harmonisation programme requirements. Blood samples were collected in the morning after an overnight fast, using a standardised venepuncture technique. VD status was classified according to the Endocrine Society guidelines and the Central European countries consensus: deficiency - 25(OH)D <30 nmol/L; insufficiency - 30.0-50.0 nmol/L; suboptimal - 50.0-75.0 nmol/L; optimal - ≥ 75.0 nmol/L [2].

Dietary Assessment. Nutritional status was evaluated using a combination of methods: a Food Frequency Questionnaire (FFQ) to capture general eating habits, and a quantitative 24-hour dietary recall (24-HR). The FFQ employed a six-point scale to assess the frequency of food consumption. The 24-HR was completed over three consecutive weekdays and one weekend day. For children under 10 years of age, questionnaires were completed by parents or guardians.

Statistical Analysis. Statistical processing was carried out using SPSS Statistics and R software (version ≥ 4.0). Normality of distribution was tested using the S. S. Shapiro - M. Wilk criterion. Quantitative data are presented either as median with interquartile range (Me [Q1; Q3]) or as mean $\pm$ standard deviation (M $\pm$ SD), depending on the distribution characteristics. Comparisons between two independent groups were made using the H. Mann - D. R. Whitney U test, while comparisons involving three or more groups used the W. Kruskal - W. A. Wallis test, followed by post hoc pairwise comparisons with O. J. Dunn's correction for multiple testing. Associations between 25(OH)D levels and anthropometric Z-scores were quantified using Ch. Spearman's rank correlation coefficient (r). ROC analysis was performed to determine the area under the curve (AUC), sensitivity (Se), specificity (Sp), and their respective 95.0% confidence intervals for each potential threshold. The optimal cut-off was identified using V. Youden's index ($J = Se + Sp - 1$). Statistical significance was set at $p < 0.05$.

Ethical Considerations. The study was approved by the institutional ethics committee. All participants or their legal guardians provided written informed consent. The research complied with the provisions of the Declaration of Helsinki (1975 and revised in 1983).

Results and discussion. In the overall sample ($n = 562$), the median serum 25(OH)D concentration was 41.7 nmol/L (first quartile [Q1]: 27.3; third quartile [Q3]: 57.2 nmol/L). VD deficiency (25(OH)D <30.0 nmol/L) was found in 31.3 % of children, insufficiency (30.0-50.0 nmol/L) in 40.6 %, suboptimal levels (50.0-75.0 nmol/L) in 22.1 %, and optimal levels (≥ 75.0 nmol/L) in only 6.0 %. Thus, reduced VD status (<50.0 nmol/L) was observed in 71.9 % of the children examined. Among adolescents aged 15-17 years, this proportion reached 81.2 %, compared with 61.4 % in children aged 3-6 years, reflecting a clear age-related decline in VD status. Data from the large reference laboratory dataset for 2022-2025 ($n \approx 70,000$) showed that the proportion of reduced status

ranged from 53.5 % to 61.9 % in children aged 5-8 years, 68.3-71.1 % in those aged 9-12 years, and 77.7-77.8 % in adolescents aged 13-16 years, confirming the external validity of the main study sample findings.

Anthropometric characterisation of the sample revealed a clear age-related trajectory (one-way ANOVA / W. Kruskal - W. A. Wallis test, $p < 0.001$ for all parameters): mean body weight increased from 19.5 ± 2.3 kg in the youngest group (3-6 years) to 55.5 ± 11.1 kg in adolescents aged 15-17 years, height from 113.9 ± 4.5 cm to 171.0 ± 6.6 cm, and BMI from 15.0 ± 1.2 kg/m² to 18.8 ± 2.8 kg/m². According to the WHO 2007 growth reference Z-score criteria, normal BMI (-1 standard deviation (SD) $\leq$ BMI $\leq$ $+1$ SD) was observed in 57.1 % of children ($n = 321$). Overweight and obesity (BMI $> +1$ SD) were present in 21.7 % ($n = 122$; 95.0% CI: 18.4-25.4 %), of whom 8.5 % ($n = 48$) were classified as obese (BMI $> +2$ SD). Underweight (BMI < -1 SD) was found in 21.2 % ($n = 119$; 95.0% CI: 17.9-24.8 %). This coexistence of excess and deficient nutritional status - referred to in the contemporary literature as the "double burden of malnutrition" - is characteristic of regions with transitional economies.

Ch. Spearman's rank correlation analysis revealed statistically significant negative associations between 25(OH)D levels and all major anthropometric parameters: BMI ($r = -0.090$, $p = 0.032$), skinfold thickness ($r = -0.156$, $p < 0.001$), body weight ($r = -0.169$, $p < 0.001$), and height ($r = -0.177$, $p < 0.001$). It should be noted that the negative correlation with height and body weight partially reflects age-related characteristics: older children have higher anthropometric values and, simultaneously, lower VD levels due to reduced time spent outdoors and decreased physical activity.

Stratified analysis by BMI category yielded more specific findings. In the group of children with overweight and obesity (BMI $> +1$ SD, $n = 122$), the mean 25(OH)D level was 37.4 ± 17.9 nmol/L, which was significantly lower than in children with normal BMI (42.3 ± 19.0 nmol/L; $p = 0.027$, H. Mann - D. R. Whitney U test). The proportion of reduced VD status in the overweight group reached 82.8 % (deficiency <30.0 nmol/L: 38.5 %; insufficiency 30.0-50.0 nmol/L: 44.3 %), compared with 69.2 % in the normal-weight group and 68.1 % in the underweight group. Among adolescents aged 15-17 years with overweight, the mean 25(OH)D level fell to approximately 34.8 nmol/L, and the proportion of reduced status reached 90.0 %, suggesting a cumulative effect of obesity and pubertal decline in physical activity.

The mechanism underlying the inverse relationship between adipose tissue stores and 25(OH)D levels is well established. First, VD is fat-soluble and is actively sequestered in adipose tissue; as total adipose mass increases, a "volumetric dilution" effect occurs, reducing circulating 25(OH)D concentrations. Second, $1,25(\text{OH})_2\text{D}$, acting through the VD receptor (VDR) in preadipocytes, inhibits adipocyte differentiation by suppressing PPAR γ ; in VD deficiency, this inhibitory mechanism is weakened, leading to fat accumulation. Third, elevated parathyroid

hormone (PTH) in the setting of VD deficiency enhances Ca^{2+} influx into adipocytes, promotes lipogenesis, and suppresses lipolysis. The bidirectional nature of this causal relationship has been confirmed by Mendelian randomisation studies: obesity has been shown to significantly lower VD levels, whereas the reverse direction - VD deficiency leading to obesity - is less well supported by evidence, though it remains pathogenetically plausible.

ROC analysis demonstrated that the 25(OH)D level was a statistically significant predictor for three anthropometric endpoints (Table 1).

Table 1

Diagnostic accuracy of serum 25-hydroxyvitamin D concentration as a predictor of anthropometric deviations in children aged 3-17 years in southern Ukraine: results of ROC analysis (n = 562) (area under the curve, predictive threshold, sensitivity, specificity, V. Youden's index)

Anthropometric indicator	AUC (95% CI)	25(OH)D threshold, nmol/L	Se, %	Sp, %	V. Youden's index
BMI-for-age $>+1$ SD	0.70 (0.65-0.75)	50.0	68.0	66.0	0.34
HFA <-1 SD	0.65 (0.59-0.71)	42.0	63.0	62.0	0.25
WHtR >0.50	0.68 (0.62-0.73)	47.0	65.0	64.0	0.29

Notes: AUC - area under the ROC curve; Se - sensitivity; Sp - specificity; CI - confidence interval. The optimal threshold was determined using V. Youden's index ($J = \text{Se} + \text{Sp} - 1$).

For BMI-for-age $>+1$ SD, the AUC was 0.70 (95.0% CI: 0.65-0.75; $p < 0.001$), indicating fair discriminatory ability. The optimal 25(OH)D threshold for identifying the risk of overweight, determined by maximising V. Youden's index ($J = 0.34$), was 50.0 nmol/L: Se 68.0 %, Sp 66.0 %. This means that at a 25(OH)D level below 50.0 nmol/L, 68.0 % of children with overweight will be correctly identified as being at risk, while at a level of 50.0 nmol/L or above, 66.0 % of children with normal weight will be correctly excluded from risk. This derived threshold of 50.0 nmol/L aligns with the recommended minimum target 25(OH)D level for children according to the Central European countries consensus [7], underscoring its clinical relevance.

For HFA <-1 SD (risk of linear growth retardation), the AUC was 0.65 (95.0% CI: 0.59-0.71; $p < 0.001$), with an optimal threshold of 42.0 nmol/L ($J = 0.25$; Se = 63.0 %, Sp = 62.0 %). The lower threshold for growth retardation compared with that for overweight risk is consistent with published data indicating that the critical 25(OH)D level required to support normal bone mineralisation and linear growth is lower than that required for effects on adipose metabolism [4].

For WHtR >0.50 (central obesity), the AUC was 0.68 (95.0% CI: 0.62-0.73), with an optimal threshold of 47.0 nmol/L ($J = 0.29$; Se = 65.0 %, Sp = 64.0 %). This intermediate position of WHtR between BMI-for-age and

HFA accords with the dual pathogenetic role of VD in both fat deposition and other metabolic processes [7].

These findings are in good agreement with the results of other studies. A. M. Shulhai et al. [11] showed that in adolescents with obesity, VD deficiency and insufficiency are associated with a higher prevalence of metabolic syndrome components and insulin resistance, with the 50.0 nmol/L threshold being clinically meaningful for risk stratification. Y. Xu et al. [13] and Z. Hajhashemy et al. [4] demonstrated that children and adolescents with lower 25(OH)D levels have higher BMI, waist circumference, and WHtR values, with the lowest VD quintile/decile characterised by the greatest anthropometric deviations. In a consensus paper for Central and Eastern Europe, the proportion of children and adolescents with 25(OH)D <50.0 nmol/L was approximately 65.0-70.0 %, and the recommended target level for healthy children was set at no less than 50.0 nmol/L [7]. Recent multicentre studies from Ukraine also show a high prevalence of reduced VD status in the paediatric population, highlighting the representativeness of our findings for the national context [3, 10]. The 50.0 nmol/L threshold we have established for overweight risk fully aligns with these recommendations.

From a pathophysiological perspective, low VD status combined with excess body weight may create a vicious pathological cycle: obesity $\rightarrow$ sequestration of VD in adipose tissue $\rightarrow$ reduced 25(OH)D level $\rightarrow$ weakened VDR-dependent inhibition of adipogenesis $\rightarrow$ further adipose tissue accumulation $\rightarrow$ worsening obesity [1, 4]. Breaking this cycle requires a combined approach: correction of VD status alongside normalisation of body weight. Results from genetic studies using Mendelian randomisation further support the notion that increased fat mass is causally associated with lower 25(OH)D levels, while the reverse direction (VD deficiency leading to obesity) appears weaker though still pathogenetically plausible [4].

Regarding linear growth, the importance of maintaining 25(OH)D levels above our identified threshold of 42.0 nmol/L is consistent with studies on the role of VD in regulating the somatotropin - IGF-1 axis, in which VDR-mediated mechanisms promote optimal insulin-like growth factor 1 (IGF-1) activity and bone mineralisation [5]. The clinical link between VD status and linear growth is particularly pronounced in at-risk groups: among children with idiopathic short stature, the prevalence of VD deficiency and insufficiency approaches half of all cases, which correlates with our findings regarding HFA <-1 SD [2].

Several limitations of this study should be acknowledged. The cross-sectional design precludes the determination of causal relationships. The sample is drawn from a single region. Physical activity levels and duration of sun exposure were not included as covariates in the ROC analysis. Prospective cohort studies, as well as interventional trials involving correction of VD status with subsequent monitoring of anthropometric endpoints, are needed to verify the predictive thresholds identified here.

Conclusions. In 562 children aged 3-17 years from the southern region of Ukraine, serum 25-hydroxyvitamin D levels were statistically significantly associated with physical development parameters, including body mass index, skinfold thickness, and body weight. Reduced 25-hydroxyvitamin D levels below 50.0 nmol/L were found in 82.8 % of children with overweight and in 68.1 % of underweight children, confirming the phenomenon of double burden of malnutrition.

Receiver operating characteristic analysis demonstrated the predictive value of 25-hydroxyvitamin D for identifying deviations in anthropometric parameters, with the area under the curve reaching 0.70 for overweight, 0.68 for central obesity, and 0.65 for linear growth retardation.

Differentiated predictive thresholds were established: 50.0 nmol/L for the risk of overweight, 47.0 nmol/L for the risk of central obesity, and 42.0 nmol/L for the risk of linear growth retardation.

These findings confirm that the study aim was achieved: an association between 25-hydroxyvitamin D levels and physical development indicators in children has been established, and predictive thresholds for identifying the risk of anthropometric deviations have been determined. The derived thresholds can serve as practical criteria for screening vitamin D deficiency in children in the southern region of Ukraine. Causal relationships require further verification in prospective cohort studies.

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Authors' contribution. 1. Concept and definition of the research design - V. Babienko. 2. Collection of study material - A. Rozhnova. 3. Processing of study material - A. Rozhnova. 4. Writing the manuscript - A. Rozhnova. 5. Editing and reviewing the manuscript - V. Babienko. 6. Project administration - V. Babienko.

Serum 25-Hydroxyvitamin D Levels in Children Aged 3-17 Years in Southern Region of Ukraine and Their Association with Anthropometric Z-Scores: Establishing a Predictive Threshold

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Introduction. Introduction. Vitamin D deficiency is common in children and may affect physical development. In Southern Ukraine, reduced serum 25-hydroxyvitamin D (25(OH)D) and a double burden of malnutrition (underweight and overweight / obesity) highlight the need for clinically relevant 25(OH)D thresholds in paediatrics.

The aim of the study. To assess the association between serum 25-hydroxyvitamin D and anthropometric z-scores in children aged 3-17 years and to determine prognostic thresholds for growth deviations using ROC analysis.

Materials and methods. A cross-sectional study included 562 children (3-17 years) examined in paediatric facilities in Southern Region of Ukraine. Serum 25(OH)D was measured by electrochemiluminescence immunoassay; physical development was assessed using WHO height-for-age (HFA), weight-for-age (WFA), BMI-for-age and waist-to-height ratio (WHtR) z-scores. Vitamin D status was classified as deficiency <30, insufficiency 30.0-50.0, suboptimal 50.0-75.0 and optimal ≥ 75.0 nmol/L. Ch. Spearman's correlation and ROC analysis (AUC, sensitivity, specificity, V. Youden's index) were applied.

Results. Median 25(OH)D was 41.7 nmol/L; 71.9 % of children had 25(OH)D <50.0 nmol/L. Serum 25(OH)D correlated negatively with BMI, skinfold thickness and body weight; 82.8 % of children with overweight / obesity had reduced vitamin D status. ROC analysis showed AUC 0.70 for BMI-for-age > +1 SD, 0.68 for WHtR >0.50 and 0.65 for HFA < -1 SD. Optimal thresholds were 50.0, 47.0 and 42.0 nmol/L for excess body weight, central obesity and linear growth impairment, respectively.

Conclusions. Serum 25-hydroxyvitamin D is associated with anthropometric deviations in children aged 3-17 years; the identified thresholds may support risk-oriented screening and prevention of growth disturbances related to vitamin D deficiency.

Keywords: vitamin D, 25-hydroxyvitamin D, children, physical development, ROC analysis, BMI-for-age, central obesity, growth retardation.

Уміст 25-гідроксिवітаміну D в сироватці крові дітей 3–17 років Південного регіону України та його зв'язок із антропометричними Z-показниками: визначення прогнозного порогу

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Вступ. Дефіцит вітаміну D поширений у дитячій популяції і може впливати на фізичний розвиток. Для Південного регіону України зафіксовано велику частоту зниженого показника 25-гідроксिवітаміну D (25(OH)D) в сироватці крові (<50,0 нмоль/л) серед дітей і підлітків, особливо віком 15–17 років. Майже однакова частка дефіциту маси тіла (21,2 %) і надмірної маси / ожиріння (21,7 %) за критеріями індексу маси тіла (ІМТ) для віку відображає подвійне нутритивне навантаження, свідчить про складну взаємодію між харчовим статусом і метаболізмом вітаміну D. Це обґрунтовує потребу визначити клінічно значущі порогові значення 25(OH)D у педіатричній практиці.

Мета. Оцінити зв'язок між показником 25(OH)D в сироватці крові й антропометричними Z-показниками у дітей віком 3–17 років і з'ясувати прогнозний поріг відхилень фізичного розвитку за допомогою ROC-аналізу.

Матеріали й методи. До одноцентрового поперечного дослідження включено 562 дитини віком 3–17 років (51,4 % – хлопчики, 48,6 % – дівчатка), яких обстежували у педіатричних закладах Південного регіону України. Уміст 25(OH)D в сироватці крові визначали методом електрохемілюмінесцентного імунологічного аналізу. Фізичний розвиток оцінювали за Z-показниками Всесвітньої організації охорони здоров'я: зріст для віку (height-for-age - HFA), маса тіла для віку (weight-for-age - WFA), індекс маси тіла для віку (BMI-for-age) та відношення обводу талії до зросту (waist-to-height ratio - WHtR). Статус вітаміну D класифікували як дефіцит <30,0 нмоль/л, недостатність 30,0–50,0 нмоль/л, субоптимальний показник 50,0–75,0 нмоль/л, опти-

мальний – $\geq 75,0$ нмоль/л. Для статистичного аналізу застосовували кореляцію Ч. Спірмена й ROC-аналіз із визначенням AUC, чутливості, специфічності, індексу В. Юдена ($J = Se + Sp - 1$); $p < 0,05$ вважали статистично значущим.

Результати. Медіана 25(OH)D становила 41,7 нмоль/л (Q1 27,3; Q3 57,2), при цьому в 71,9 % дітей показник 25(OH)D був $< 50,0$ нмоль/л. Визначено від'ємні кореляції між 25(OH)D та ІМТ ($r = -0,090$; $p = 0,032$), товщиною шкірно-жирової складки ($r = -0,156$; $p < 0,001$) і масою тіла ($r = -0,169$; $p < 0,001$). У дітей із надмірною масою тіла / ожирінням ($n = 122$) середній показник 25(OH)D був нижчим, ніж у дітей із нормальним ІМТ ($37,4 \pm 17,9$ проти $42,3 \pm 19,0$ нмоль/л; $p = 0,027$), а знижений статус вітаміну D ($< 50,0$ нмоль/л) фіксували у 82,8 %. ROC-аналіз показав AUC 0,70 (95,0% ДІ 0,65–0,75) для BMI-for-age $> +1$ SD, 0,68 (95,0% ДІ 0,62–0,73) для WHtR $> 0,50$ і 0,65 (95,0% ДІ 0,59–0,71) для HFA < -1 SD (усі $p < 0,001$). Оптимальний поріг 25(OH)D становив 50,0 нмоль/л для ризику надлишкової маси тіла, 47,0 нмоль/л – для центрального ожиріння, 42,0 нмоль/л – для ризику порушення лінійного росту.

Висновки. Показник 25(OH)D в сироватці крові асоціюється з антропометричними відхиленнями у дітей віком 3–17 років Південного регіону України та демонструє прийнятну прогностичну здатність щодо надлишкової маси тіла, центрального ожиріння й затримки лінійного росту. Виявлені порогові значення 50,0, 47,0 і 42,0 нмоль/л можуть бути використані для ризикорієнтованого скринінгу та профілактики порушень фізичного розвитку, асоційованих із дефіцитом вітаміну D, в педіатричній практиці.

Ключові слова: вітамін D, 25-гідроксивітамін D, діти, фізичний розвиток, ROC-аналіз, індекс маси тіла для віку, центральне ожиріння, затримка росту.

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