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TRAJECTORIES OF SERUM 25(OH)D IN CHILDREN WITH PARALYTIC SYNDROMES WHEN USING VITAMIN D FROM DIFFERENT MANUFACTURERS AND IN DIFFERENT DOSES

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Introduction

The prevalence of vitamin D deficiency and insufficiency varies significantly in different subpopulations of children depending on age and diseases, and recommendations for their correction in children with paralytic syndromes are limited.

Aim. Empirical determination of blood 25(OH)D trajectory in children with paralytic syndromes when using vitamin D from different manufacturers and in different doses.

Material and methods. The 25(OH)D (ng/ml) of blood serum was analyzed by immune-enzymatic method "Labline-90" (Austria) with the test system "Monobind Inc." (ELISA, USA) in 77 children with paralytic syndromes aged 1-18 years, a repeat study after vitamin D3 supplementation was conducted in 36 children. The rate of increase in 25(OH)D concentration per month was calculated. Methods of descriptive statistics, non-parametric correlation analysis and Kaplan-Meier survival analysis were used with MedCalc Statistical Software (Belgium).

This study was approved by the Ethics Committee (protocol No. 5, October 2021), which was conducted with the involvement of minor patients and did not contain measures that could harm their health.

The research was carried out within the framework of the Department of Pediatrics of Kharkiv National Medical University "Medical and social aspects of adaptation of children with somatic pathology in modern conditions" (state registration number 0120U102471, 2020).

Results. Vitamin D insufficiency was diagnosed in 17% of children with paralytic syndromes, and vitamin D deficiency in 73%, so daily doses of 2000-4000 IU of vitamin D3 from different manufacturers were recommended at the discretion of the parents for 6 months. In reality, children received doses from 500 to 5000 IU randomly, from 2 to 7 months. Doses were stratified as greater than 2000 IU and less than 2000 IU. If the child received a dose of 2000 IU or more, the rate of increase of 25(OH)D in the blood in children was 3.6 ng/ml per month, if the dose was less than 2000 IU - 1.6 ng/ml per month.

Conclusions. Children with paralytic syndromes should be screened and monitored for serum 25(OH)D levels. With a serum 25(OH)D level of less than 20 ng/ml, daily administration of vitamin D3 in a dose of at least 2000 IU for at least 6 months allows reaching a 25(OH)D level of 30 ng/ml in most of them. Further large-scale studies are needed to supplement current recommendations for vitamin D3 supplementation in children with paralytic syndromes.

Key words: Children; Paralytic Syndromes; 25-hydroxyvitamin D, Vitamin D.

Introduction

Epidemiologic studies of vitamin D deficiency in the pediatric population have shown that the prevalence of 25-hydroxyvitamin D (25(OH)D) < 20 ng/ml in the blood serum is about 15%, and that of severe deficiency < 10 ng/ml is 1-2%. However, the prevalence of vitamin D deficiency and insufficiency varies considerably from one country to another and in different subpopulations of children depending on age and comorbidities [1, 2]. Known risk factors for vitamin D insufficiency and deficiency include reduced sun exposure, overweight or obesity, anticonvulsant medication, malnutrition or malabsorption, and other gastrointestinal diseases [3-9].

According to the recommendations of the Endocrine Society "Evaluation, Treatment, and Prevention of Vitamin D Deficiency: an Endocrine Society Clinical Practice Guideline" (2011). the following risk groups are identified: infants who are exclusively breastfed or premature infants; dark-skinned infants; children with low vitamin D intake; obese children; infants and young children with growth disorders; children taking medications that contribute to vitamin D deficiency, including certain anticonvulsants, antiretroviral drugs or glucocorticoids; children with chronic diseases associated with impaired absorption, such as celiac disease, cystic fibrosis, inflammatory bowel disease, or cholestatic liver disease; children with malnutrition, amenorrhea, or immobilization;

children with chronic kidney disease or severe liver dysfunction, pregnant adolescents, or lactating adolescents [10].

That is, children with paralytic syndromes are potentially at risk of vitamin D deficiency and insufficiency, but screening in this category of children is not recommended at the national level, as well as the reduced awareness of primary care physicians in this area [11, 12].

The aim of the study

To empirically determine the paths of blood 25(OH)D in children with paralytic syndromes when using vitamin D from different manufacturers and in different doses.

Material and methods of the study

The study design was single-center, cross-sectional: the period from October 2021 to March 2022 (autumn-spring season). Demographic and clinical data were evaluated, and levels of motor dysfunction were determined [13].

The study involved 77 children with paralytic syndromes. Inclusion criteria: children aged 1-18 years with paralytic syndromes according to ICD-10 (cerebral palsy G 80, hemiplegia G 81, paraplegia and tetraplegia G 82, other paralytic syndromes G 83) associated with CNS damage due to hypoxia, bleeding, thrombosis, trauma; congenital brain defects. Exclusion criteria: rickets-like hereditary

diseases, undiagnosed progressive conditions with central nervous system disorders of unclear etiology, rickets in young children, congenital or hereditary skeletal disorders, liver and kidney disease, and patients who have already taken synthetic vitamin D supplements.

Serum 25(OH)D (ng/ml) was determined twice by enzyme-linked immunosorbent assay on a Labline-90 analyzer (Austria) using a commercial test system manufactured by MonobindInc. (ELISA, USA) according to the instructions.

The first serum 25(OH)D determination was performed in 77 children, and the second determination was performed in 36 children after vitamin D3 supplementation in the time interval of 2-7 months (median 6 months). Vitamin D3 supplementation was prescribed in any way available to parents (availability in pharmacies, liquid forms for chewing and swallowing dysfunction, financial capacity, etc.) in doses of 2000 - 4000 IU. After 6 months, the blood 25(OH)D study was repeated. Depending on the formulation, the dose was stratified as < 2000 IU and ≥ 2000 IU. An analysis was performed of an indicator independent of age and form of the drug, time - the rate of increase in 25(OH)D concentration over a certain period of time in children with paralytic syndromes, considering the initial, final level (state of deficiency and/or insufficiency) and dose of vitamin D3, which were provided by the manufacturer of a particular form of the drug, that is, to determine the empirical trajectories of its increase (or decrease) in this group of patients, with possible individual approaches to further correction according to the formula:

Rate of rise = $[25(OH)D1 - 25(OH)D2]/\text{hour}$, where:

25(OH)D1 is the serum value at the first test (ng/ml);

25(OH)D2 - blood serum level in the second study (ng/ml);

time in months.

Vitamin D insufficiency was considered at a serum 25(OH)D level of 20-30 ng/mL, and deficiency at a level < 20 ng/mL [60].

Statistical analysis was performed using MedCalcStatisticalSoftware version 18.2.1 (MedCalcSoftwarebvba, Ostend, Belgium; 2018). Descriptive analysis, frequency determination and 95% confidence interval (CI), nonparametric correlation analysis r (Spearman's rank correlation coefficient) and its 95% confidence interval (CI) were used. To provide further evidence of the time-dose-concentration relationships for the purpose of optimizing serum 25(OH)D, a series of Kaplan-Meier survival analysis procedures were performed to examine the distribution of events and to estimate conditional probabilities at each time point when an event occurs, or a covariate instead of time, and the ability to select a stratification variable to separately analyze different levels (strata) of that variable. The difference in the parameters was considered statistically significant at $p < 0.05$.

This study was approved by the Ethics Committee (Protocol No. 5 of October 2021), which was conducted with the involvement of minor patients and did not contain activities that could harm their health. Both parents were informed about the methods and scope of the study and agreed to their children's participation in this survey.

The study was conducted within the framework of the research work of the Department of Pediatrics of Kharkiv National Medical University "Medical and social aspects of adaptation of children with somatic pathology in modern conditions" (state registration number 0120U102471, 2020).

Study results and discussion. Paralytic syndromes in children were the result of cerebral palsy in 34 (44.2%), congenital brain defects in 23 (29.8%), and perinatal pathology in 20 (25.9%). The median age of the children with paralytic syndromes was 5 years, the minimum age was 1 year, and the maximum age was 17.5 years. Demographic and clinical data are presented in Table 1.

Table 1

Demographic data of children with paralytic syndromes

| Data | n, % | 95 % CI |
|-----------------------------|-----------|---------|
| Age | | |
| 1 – 4 years | 20 (25,9) | 16 – 35 |
| 4 – 7 years | 33 (42,8) | 31 – 54 |
| 7 – 11 years | 14 (18,1) | 9 – 26 |
| 11 – 18 years | 10 (12,9) | 5 – 20 |
| Males | 48 (62,4) | 51 – 72 |
| Females | 29 (37,6) | 27 – 48 |
| Rural area | 16 (20,7) | 12 – 30 |
| GMFCS | | |
| III level | 14 (18,1) | 9 – 26 |
| IV level | 19 (24,6) | 15 – 34 |
| V level | 44 (57,1) | 45 – 68 |
| Anticonvulsants | 46 (59,7) | 49 – 71 |
| First study of 25(OH)D: | | |
| Insufficiency 20 – 30 ng/ml | 13 (16,8) | 8 – 25 |
| Deficiency < 20 ng/ml | 56 (72,7) | 63 – 82 |

Despite the fact that 69 (89.6%) children with paralytic syndromes were diagnosed with hypovitaminosis D, only 36 families agreed to further study - vitamin D3 supplementation with repeated serum 25(OH)D determination. All children, depending on their condition (deficiency or

insufficiency), were recommended to receive 2000 IU or 4000 IU of vitamin D3 from any manufacturer with repeated serum 25(OH)D determination in 6 months. However, the reality was that parents changed the prescribed dose on their own and performed a repeat test at a time convenient to them, without observing

the time interval. Therefore, we performed an individual analysis for each case (Table 2).

Stratification of the rate of increase in blood 25(OH)D concentration over time according to dose

showed that the median value in children with paralytic syndromes was 3.18 ng/ml/month: for children receiving a dose < 2000 IU - 1.6 ng/ml/month, > 2000 IU - 3.6 ng/ml/month ($p=0.0260$) (Fig. 1).

Table 2

Peculiarities of taking vitamin D3 in children with paralytic syndromes

| Sing | N, (%), N=36 | 95 % CI |
|--------------------|--------------|---------|
| Dose of Vitamin D3 | | |
| 500 IU | 1 (2,7) | 0 – 13 |
| 1000 IU | 3 (8,3) | 0 - 16 |
| 1200 IU | 2 (5,5) | 0 – 14 |
| 1500 IU | 3 (8,3) | 0 - 16 |
| 2000 IU | 4 (11,1) | 0- 21 |
| 2500 IU | 2 (5,5) | 0 – 14 |
| 3000 IU | 4 (11,1) | 0 - 21 |
| 3500 IU | 6 (16,6) | 4 - 29 |
| 4000 IU | 6 (16,6) | 4 - 29 |
| 5000 IU | 4 (11,1) | 0 - 21 |
| Time Convenient | | |
| 2 month | 1 (2,7) | 0 - 13 |
| 3 month | 3 (8,3) | 0 - 16 |
| 4 month | 4 (11,1) | 0 - 21 |
| 5 month | 4 (11,1) | 0 - 21 |
| 6 month | 21 (58,3) | 41 - 74 |
| 7 month | 3 (8,3) | 0 - 16 |

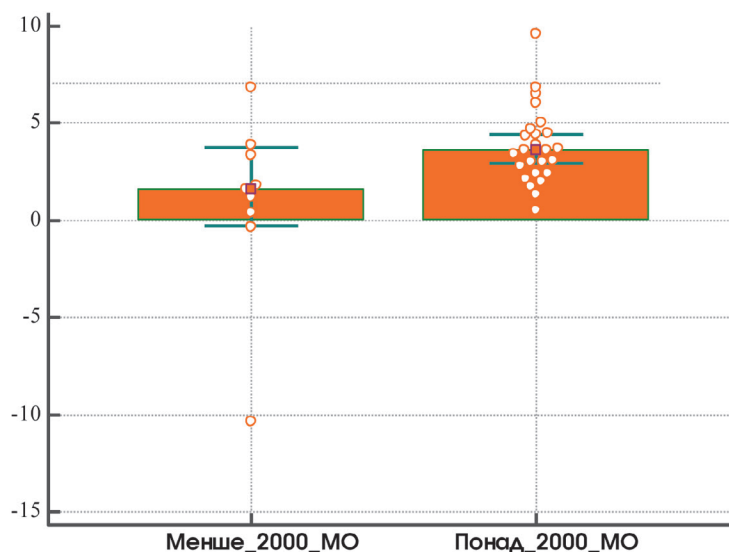


Figure 1. One-month rate of increase in serum 25(OH)D by vitamin D3 dose

In two cases we observed a decrease in serum 25(OH)D compared to the first study, i.e. a decrease in serum 25(OH)D concentration.

Example: A 5-year-old child with cerebral palsy, motor dysfunction level II. The serum 25(OH)D level at the first examination is 13.85 ng/ml (deficient state). The recommended dose was 4000 IU for 6 months with continued monitoring. However, the parents were “afraid of high doses” and prescribed 500 IU vitamin D3 for 7 months. Repeated examination of serum 25(OH)D - 11.37 ng/ml, the deficiency state deepened and decreased by (-2.48) ng/ml/month.

We found a significant correlation between the rate of increase and the dose of vitamin D3 taken ($r=0.4$, 95% CI 0.0580 - 0.629, $p=0.0225$).

To provide further evidence of the time-dose-concentration relationships, a series of Kaplan-Meier survival analyses were performed to optimize serum 25(OH)D. It was shown that the lower the dose of vitamin D3, the longer the prescribed intake (at least 6 months) (Chi-squared test - 10.7, $p=0.05$). It was proved that to correct vitamin D deficiency or insufficiency in children with paralytic syndromes from 1 to 18 years of age, a “stratified dose” of vitamin D32000 IU should be prescribed and administered daily for at least 6 months, according to the cumulative frequency of the rate of increase (Chi-square test - 42.6, $p=0.0001$).

The frequency of serum 25(OH)D deficiency and insufficiency in the first (1) and second (2) study was compared: deficiency in children with paralytic syndromes was 73% (1) and 11% (2) ($p=0.0001$),

insufficiency 17% (1) and 28% (2) ($p=0.1728$).

In recent decades, an increasing number of studies have demonstrated the multifunctional effect of vitamin D on the functioning of organs and systems, as vitamin D can also regulate many other cellular functions [14-20].

It is known that one of the most important international recommendations for vitamin D3 intake in children and adults today is the Endocrine Society Clinical Practice Guideline "Evaluation, Treatment, and Prevention of Vitamin D Deficiency: an Endocrine Society Clinical Practice Guideline" (2011), which recommends that children aged 1-18 years at risk of vitamin D deficiency receive 2000 IU/day (upper limit 4000 IU) of vitamin D2 or vitamin D3 for at least 6 weeks or 50,000 IU of vitamin D2 once weekly for at least 6 weeks to achieve a blood 25(OH)D level above 30 ng/ml, followed by maintenance therapy of 600-1000 IU/ml. Children with neurological disorders are not included in the risk group [10]. In contrast to these recommendations, in the European Society for Paediatric Gastroenterology, Hepatology and Nutrition Guidelines for the Evaluation and Treatment of Gastrointestinal and Nutritional Complications in Children with Neurological Impairment (2017), the working group recommends the assessment of micronutrient status (e. g. vitamin D, iron, calcium, phosphorus status) as part of the nutritional assessment of children with neurological disorders because monitoring micronutrient status in children with neurological disorders can have a significant impact on nutritional adequacy, hospital costs, and future outcomes. However, the doses and duration of correction are not specified [21].

We have shown that daily doses of less than 2000 IU of vitamin D3 and its administration for less than 6 months do not reach the level of 30 ng/ml in children aged 1-18 years with paralytic syndromes.

The data obtained provide grounds for optimizing approaches to correct hypovitaminosis D, which makes it important to improve the quality of life of such children. An important component is screening and monitoring in children and timely medical care. It should be noted that screening, monitoring, and vitamin D supplementation prevent the development of more serious complications,

such as osteopenia and fractures, secondary immunodeficiency, cognitive impairment, and other conditions associated with hypovitaminosis D. Therefore, the timely initiation of vitamin D3 supplementation in children with paralytic syndromes theoretically has social, medical, and economic implications [22-25].

Conclusions

Children with paralytic syndromes should be screened and monitored for serum 25(OH)D levels, as 17% have vitamin D insufficiency and 73% have deficiency. The trajectory of increase or decrease in 25(OH)D levels in children with paralytic syndromes depends on the dose and timing of vitamin D3 supplementation. If the serum 25(OH)D level is less than 20 ng/mL, daily administration of vitamin D3 at a dose of at least 2000 IU for at least 6 months allows most of them to reach a 25(OH)D level of 30 ng/mL. The average rate of increase of 25(OH)D concentration in ng/ml in 1 month in children aged 1-18 years with paralytic syndromes is 1.6 ng/ml/month if children receive a dose of vitamin D3 less than 2000 IU, if children receive a dose of 2000 IU or more - 3.6 ng/ml/month, which should be considered in the medical monitoring of this category of patients. Communication with parents of children with paralytic syndromes is needed regarding dosages, forms, and monitoring of adequate serum 25(OH)D levels. Further large-scale studies are needed to supplement current recommendations for vitamin D3 supplementation in children with paralytic syndromes.

Prospects for further research: determination of 25(OH)D concentration in children with paralytic syndromes depending on: 1) the type of diet and the use of clinical formulations with a specific vitamin D3 content; 2) the lifestyle and duration of sun exposure; 3) different anticonvulsant therapy regimens.

Conflict of interest. The authors declare no conflict of interest.

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References:

- Mansbach JM, Ginde AA, Camargo CA Jr. Serum 25-hydroxyvitamin D levels among US children aged 1 to 11 years: do children need more vitamin D? *Pediatrics*. 2009;124(5):1404-10. doi: 10.1542/peds.2008-2041
- Cashman KD, Dowling KG, Škrabáková Z, Gonzalez-Gross M, Valtueña J, De Henauw S, et al. Vitamin D deficiency in Europe: pandemic? *Am J Clin Nutr*. 2016;103(4):1033-44. doi: 10.3945/ajcn.115.120873
- Misra M, Pacaud D, Petryk A, Collett-Solberg PF, Kappy M; Drug and Therapeutics Committee of the Lawson Wilkins Pediatric Endocrine Society. Vitamin D deficiency in children and its management: review of current knowledge and recommendations. *Pediatrics*. 2008;122(2):398-417. doi: 10.1542/peds.2007-1894
- Shulhai AM, Pavlyshyn H, Shulhai O. Peculiarities of the prevalence and risk factors for vitamin D deficiency in overweight and obese adolescents in Ukraine. *Archives of the Balkan Medical Union*. 2019;54(1):57-63. doi: 10.31688/ABMU.2019.54.1.08
- Harel Z, Flanagan P, Forcier M, Harel D. Low vitamin D status among obese adolescents: prevalence and response to treatment. *J Adolesc Health*. 2011;48(5):448-52. doi: 10.1016/j.jadohealth.2011.01.011
- Vijayakumar M, Bk A, George B, Bhatia V. Vitamin D Status in Children on Anticonvulsant Therapy. *Indian J Pediatr*. 2022;89(6):541-5. doi: 10.1007/s12098-021-03853-y
- Sudfeld CR, Mugusi F, Muhimi A, Aboud S, Nagu TJ, Ulenga N, et al. Efficacy of vitamin D3 supplementation for the prevention of pulmonary tuberculosis and mortality in HIV: a randomised, double-blind, placebo-controlled trial. *Lancet HIV* [Internet]. 2020[cited 2023 May 15];7(7):e463-71. Available from: [https://www.thelancet.com/journals/lanhiv/article/PIIS2352-3018\(20\)30108-9/fulltext](https://www.thelancet.com/journals/lanhiv/article/PIIS2352-3018(20)30108-9/fulltext) doi: 10.1016/S2352-3018(20)30108-9
- Chau YY, Kumar J. Vitamin D in chronic kidney disease. *Indian J Pediatr*. 2012;79(8):1062-8. doi: 10.1007/s12098-012-0765-1
- Polzonetti V, Pucciarelli S, Vincenzetti S, Polidori P. Dietary Intake of Vitamin D from Dairy Products Reduces the Risk of Osteoporosis. *Nutrients* [Internet]. 2020[cited 2023 May 15];12(6):1743. Available from: <https://www.mdpi.com/2072-6643/12/6/1743> doi: 10.3390/nu12061743
- Holick MF, Binkley NC, Bischoff-Ferrari HA, Gordon CM, Hanley DA, Heaney RP, et al. Evaluation, treatment, and prevention of vitamin D deficiency: an Endocrine Society clinical practice guideline. *J Clin Endocrinol Metab*. 2011;96(7):1911-30. doi: 10.1210/jc.2011-0385
- Paker N, Yavuz Mollavelioglu T, Bugdaycı D, Ones K, Bardak AN, Karacan I, et al. Vitamin D levels in children with cerebral palsy. *J Pediatr Rehabil Med*. 2023;16(1):163-9. doi: 10.3233/PRM-190622
- Le Roy C, Barja S, Sepúlveda C, Guzmán ML, Olivarez M, Figueroa MJ, et al. Vitamin D and iron deficiencies in children and adolescents with cerebral palsy. *Neurologia*. 2021;36(2):112-8. doi: 10.1016/j.nrl.2017.11.005
- Palisano R, Rosenbaum P, Walter S, Russell D, Wood E, Galuppi B. Development and reliability of a system to classify gross motor function in children with cerebral palsy. *Dev Med Child Neurol*. 1997;39(4):214-23. doi: 10.1111/j.1469-8749.1997.tb07414.x
- Matías-Guío J, Oreja-Guevara C, Matias-Guío JA, Gomez-Pinedo U. Vitamin D and remyelination in multiple sclerosis.

Neurologia. 2018;33(3):177-86. doi: 10.1016/j.nrl.2016.05.001

15. Sacerdote A, Dave P, Lokshin V, Bahtiyar G. Type 2 Diabetes Mellitus, Insulin Resistance, and Vitamin D. *Curr Diab Rep* [Internet]. 2019[cited 2023 May 15];19(10):101. Available from: <https://link.springer.com/article/10.1007/s11892-019-1201-y> doi: 10.1007/s11892-019-1201-y

16. Korkmaz FN, Ozen G, Unal AU, Odabasi A, Can M, Asıcıoglu E, et al. Vitamin D levels in patients with small and medium vessel vasculitis. *Reumatol Clin*. 2022;18(3):141-6. doi: 10.1016/j.reuma.2020.11.004

17. Vernia F, Valvano M, Longo S, Cesaro N, Viscido A, Latella G. Vitamin D in Inflammatory Bowel Diseases. Mechanisms of Action and Therapeutic Implications. *Nutrients* [Internet]. 2022[cited 2023 May 15];14(2):269. Available from: <https://www.mdpi.com/2072-6643/14/2/269> doi: 10.3390/nu14020269

18. Radulović Ž, Zupan ZP, Tomazini A, Varda NM. Vitamin D in pediatric patients with obesity and arterial hypertension. *Sci Rep* [Internet]. 2021[cited 2023 May 15];11(1):19591. Available from: <https://www.nature.com/articles/s41598-021-98993-8.pdf> doi: 10.1038/s41598-021-98993-8

19. Gao YX, Zhang J, Man Q, Li Y, Jia S. The association between vitamin D levels and metabolic syndrome components among metropolitan adolescent population. *J Pediatr Endocrinol Metab*. 2021;35(1):55-63. doi: 10.1515/jpem-2021-0581

20. Libuda L, Timmesfeld N, Antel J, Hirtz R, Bauer J, Führer D, et al. Effect of vitamin D deficiency on depressive symptoms in child and adolescent psychiatric patients: results of a randomized controlled trial. *Eur J Nutr*. 2020;59(8):3415-24. doi: 10.1007/s00394-020-02176-6

21. Romano C, van Wynckel M, Hulst J, Broekaert I, Bronsky J, Dall'Oglio L, et al. European Society for Paediatric Gastroenterology, Hepatology and Nutrition Guidelines for the Evaluation and Treatment of Gastrointestinal and Nutritional Complications in Children With Neurological Impairment. *J Pediatr Gastroenterol Nutr*. 2017;65(2):242-64. doi: 10.1097/MPG.0000000000001646

22. Głąbska D, Kołota A, Lachowicz K, Skolmowska D, Stachoń M, Guzek D. The Influence of Vitamin D Intake and Status on Mental Health in Children: A Systematic Review. *Nutrients* [Internet]. 2021[cited 2023 May 15];13(3):952. Available from: <https://www.mdpi.com/2072-6643/13/3/952> doi: 10.3390/nu13030952

23. Uchitomi R, Oyabu M, Kamei Y. Vitamin D and Sarcopenia: Potential of Vitamin D Supplementation in Sarcopenia Prevention and Treatment. *Nutrients* [Internet]. 2020[cited 2023 May 15];12(10):3189. Available from: <https://www.mdpi.com/2072-6643/12/10/3189> doi: 10.3390/nu12103189

24. Alenazi KA. Vitamin D deficiency in children with cerebral palsy: A narrative review of epidemiology, contributing factors, clinical consequences and interventions. *Saudi J Biol Sci*. 2022;29(4):2007-13. doi: 10.1016/j.sjbs.2021.12.026

25. Seth A, Aneja S, Singh R, Majumdar R, Sharma N, Gopinath M. Effect of impaired ambulation and anti-epileptic drug intake on vitamin D status of children with cerebral palsy. *Paediatr Int Child Health*. 2017;37(3):193-8. doi: 10.1080/20469047.2016.1266116

ТРАЕКТОРІЇ 25(ОН)D КРОВІ У ДІТЕЙ З ПАРАЛІТИЧНИМИ СИНДРОМАМИ ПРИ ВЖИВАННІ ВІТАМІНУ D РІЗНИХ ВИРОБНИКІВ ТА У РІЗНИХ ДОЗАХ

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Резюме.

Вступ. Поширеність дефіциту та недостатності вітаміну D значно відрізняється в різних субпопуляціях дітей залежно від віку та захворювань, а рекомендацій щодо їх корекції у дітей з паралітичними синдромами обмежені.

Мета. Емпіричне визначення траєкторії 25(ОН)D крові у дітей з паралітичними синдромами при вживанні вітаміну D різних виробників та у різних дозах.

Матеріал та методи. Досліджували 25(ОН)D (нг/мл) сироватки крові імуноферментним методом «Labline-90» (Австрія) з тест-системою «MonobindInc.» (ELISA, США) у 77 дітей з паралітичними синдромами віком 1 – 18 років, повторне дослідження після дотації вітаміну D3 проведено у 36 дітей. Розраховували швидкість підйому концентрації 25(ОН)D за місяць.

Використовували методи описової статистики, непараметричного кореляційного аналізу аналізу виживання Kaplan-Meier з програмою MedCalc Statistical Software (Бельгія).

Дане дослідження схвалено Комітетом з Етики (протокол № 5 від жовтня 2021 р.), яке проводилося із залученням неповнолітніх пацієнтів і не містило заходів, які можуть завдати шкоди їх здоров'ю.

Дослідження проведено в межах НДР кафедр педіатричного профілю Харківського національного медичного університету «Медико-соціальні аспекти адаптації дітей з соматичною патологією в сучасних умовах» (номер державної реєстрації 0120U102471, 2020 рік).

Результати дослідження. У 17 % дітей з паралітичними синдромами діагностовано недостатність вітаміну D, а у 73 % - дефіцит, тому рекомендували щоденні дози 2000 – 4000 МО вітаміну D3 різних виробників на розсуд батьків упродовж 6 місяців. Реально діти отримували дози від 500 до 5000 МО хаотично, від 2 до 7 місяців. Дози стратифікували як понад 2000 МО та менше 2000 МО. Якщо дитина отримала дозу 2000 МО та більше, то швидкість підйому 25(ОН)D в крові у дітей було 3,6нг/мл на місяць, якщо дозу менше 2000 МО - 1,6 нг/мл на місяць.

Висновки. Діти з паралітичними синдромами мають підлягати скринінгу та моніторингу рівня 25(ОН)D сироватки крові. При рівні 25(ОН)D сироватки крові менше 20 нг/мл щоденне призначення вітаміну D3 у дозі не менше 2000 МО упродовж не менше ніж 6 місяців дозволяє досягти рівня 25(ОН)D 30нг/млу більшості з них. З метою доповнення сучасних рекомендацій із дотації вітаміну D3 дітям з паралітичними синдромами потрібно проведення подальших широкомасштабних досліджень.

Ключові слова: діти; паралітичні синдроми; 25-гідроксівітамін D; вітамін D.

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