Original Article

Should Vitamin D Supplementation in Cerebral Palsy Patients be a Routine Practice for Pediatricians?

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Abstract

Background: Children with cerebral palsy often develop osteopenia and rickets due to dietary deficiencies, lack of sun exposure, and sedentary lifestyle. Their bone mineral density gets drastically diminished. Therefore, this study was done in order to sensitize practicing paediatricians for routine supplementation of vitamin D in cerebral palsy even beyond infancy. **Material and Methods:** This is a Case control study. There were 50 cases of cerebral palsy aged 1-5 years qualifying the inclusion criteria (categorized as group 1) matched with normal children of same age and sex were randomly selected as control groups (categorized as group 2) after informed consent. **Results:** The prevalence of decreased vitamin D in patients with CP was 67% in cases as compared to 42% in control. Whereas the prevalence of vitamin D deficiency in study group was 21% and insufficiency was 46%. In the control group, vitamin D deficiency amounted to 4%, while insufficiency amounted to 38%. In both populations, the prevalence of insufficiency was more frequent than deficiency. The prevalence of decreased vitamin D in cases was 67% which was statistically significant, while in the control group, decreased vitamin D levels amounted to 42%. **Conclusion:** Hypovitaminosis D is very common in cerebral palsy and represents latent stage of vitamin D deficiency. Hence there is growing need for routine supplementation of vitamin D in cerebral palsy even beyond infancy.

Keywords: Cerebral Palsy, Vitamin D, deficiency, supplementation.

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Introduction

A group of abnormalities in movement and posture collectively referred to as "cerebral palsy" (CP) are believed to be brought on by non-progressive alterations in the developing or newborn brain. Along with the movement problems of cerebral palsy, symptoms of sensory, cognitive, communication, perception, and/or behaviour abnormalities are commonly present.[1] Despite generating early motor deficits, diseases related to the muscles or the peripheral nerves of the spinal cord (such as muscular dystrophies) are not regarded as cerebral palsy. Examples include spinal muscular atrophy and myelomeningocele.

It is unclear how cerebral palsy develops pathophysiologically. Events such as brain damage or aberrant brain development during the foetal, maternal, gestational, or postnatal events taking place throughout the child's neural development are to be blamed. Brain damage may happen at any moment, from pregnancy through early life. Contrary to widespread assumption, cerebral palsy occurs in less than 10% of birth-related injuries. Compared to infants born at term, children born slightly preterm (37–38 weeks) or post-term (42 weeks) had an increased incidence of cerebral palsy, according to cohort studies (40 weeks).[2] Leukomalacia infections or inflammations of the brain, basal ganglia injuries, periventricular-intraventricular haemorrhage, and hypoperfusion injuries in the middle cerebral artery distribution are only a few examples of brain damage.

According to estimates, cerebral palsy is estimated to affect 1.5–5.6 out of every 1000 live births in developing countries.[3] Preterm and extremely preterm babies are far more likely to have this disease. Males have a greater incidence than females,[4,5] (ratio of 1.33:1). Cerebral palsy risk factors might include lower socioeconomic level.[6] Mental retardation (IQ 50): 31%; seizures: 21%; and non-ambulatory: 20% are the most common comorbid conditions in children with CP.[7] The most prevalent paediatric developmental disability that lasts a lifetime is cerebral palsy (CP). It is a clinical condition defined by a persistent disturbance in motor coordination and posture brought on by non-progressive brain damage or malfunction.[8] In addition to the motor abnormalities of cerebral palsy, secondary musculoskeletal problems, seizures, and modifications in sensory, cognition, communication, and behaviour are typically present.[9] Due to aberrant muscle tone, involuntary movement,

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unstable walking, balance issues, and poor social functioning, children with CP have limits in their everyday activities, such as eating, dressing, bathing, and mobility. Additionally, they have trouble in swallowing. Malnutrition and feeding difficulties are two complications that may arise from cerebral palsy. These individuals often consume inadequate calcium and 25-hydroxyvitamin D, as well as poor general nutrition.[10]

As it encourages the digestion of dietary calcium and phosphate, 25-hydroxyvitamin D is essential for children's bone formation, mineralization, and musculoskeletal health. 25-hydroxyvitamin D also controls a wide range of cellular processes. 25-hydroxyvitamin D deficiency significantly increase the risk of metabolic syndrome, diabetes, autoimmune disorders and several cancer types. In addition to metabolic bone disease and rickets, severe 25-hydroxyvitamin D deficiency (VDD) may lead to hypocalcemia throughout a baby's and child's development.[11] Children with CP often develop osteopenia and rickets due to dietary deficiencies, lack of sun exposure, and sedentary lifestyle. The patient's bone mineral density gets drastically diminished. In addition, painful fractures are often associated with minor injuries.[12]

Aim of the study: To study the prevalence of Vitamin D deficiency and insufficiency in children with Cerebral Palsy and to compare them with normal children of same age and either sex.

Materials and Methods

The present study was conducted in the Department of Paediatrics, Career Institute of Medical Sciences and Hospital, Lucknow, U.P., from January 2021 to October 2022on children diagnosed as a case of Cerebral Palsy.

Place of study: The present study was conducted in Department of Paediatrics, Career Institute of Medical Sciences and Hospital, Lucknow, U.P. This study was approved by institutional ethical cell.ref no:CIMSH/IEC/DEC/2020/16.

Study Duration: The present one and half year study was conducted from January 2021 to October 2022.

Study Procedure: A thorough history was taken, including demographic information (age/sex), birth information. All patients had a thorough physical examination, including anthropometry, with a focus on any indicators of clinical vitamin D deficiency. Vitamin D was analysed using CLIA method after collecting 4ml blood sample in plain vial.

Study Design: This is a Case control study. There were 50 cases of cerebral palsy (categorized as group 1) matched with normal children of same age and sex were randomly selected as control groups (categorized as group 2) after informed consent.

Selection of patient

Inclusion Criteria:

Children with cerebral palsy of age group 1-5 years of either sex attending the paediatric outpatient department and inpatient care of our hospital contributed as the subjects of this study. Normal children were randomly selected as controls after informed consent.

Exclusion Criteria:

Sample Size

A total of 50 patients presenting with cerebral palsy were selected. All children satisfying inclusion criteria Controls age and sex matched with controls. Thereby 50 controls were selected.

Reference Standard (criteria)

Criteria for diagnosis of Cerebral Palsy 1

Cerebral palsy cannot be confirmed or ruled out by any conclusive testing. The diagnosis of cerebral palsy in clinical practice is often made based on observations or parent accounts of accomplished motor milestones, such as sitting, pushing oneself to a standing position, and walking, as well as an assessment of posture, deep tendon reflexes, and muscle tone.

Criteria for classification of Vitamin D deficiency (IOM)13

<12 ng/dl – Deficiency

12-20 ng/dl – Insufficiency

>20 ng/dl – Normal

>50 ng/dl – Toxicity

Results

This is a Case control study. There were 50 cases of cerebral palsy aged 1-5 years qualifying the inclusion criteria (categorized as group 1) matched with normal children of same age and sex were randomly selected as control groups (categorized as group 2) after informed consent.

Distribution of Neonatal Attributes and comparison between Control & Cases

The results based on Distribution of Neonatal Attributes and comparison between Control & Cases illustrated that mode of delivery- Elective LSCS (case=4%; control=20%) and Emergency LSCS (case=36%; control=3%) was found significant ($\chi2=6.06$; $p=0.048$) with neonatal attributes whereas all other parameters such as sex ($p=1.000$), gestation (0.372), birth weight (0.110) were all found non significant [Table 1].

Distribution of Complaints and comparison between Control & Cases

The results based on Distribution of Complaints and comparison between Control & Cases illustrated that Birth Asphyxia ($\chi2=21.95$; $p<0.001$), Developmental Delay ($\chi2=100.00$; $p<0.001$), Neonatal Seizure ($\chi2=11.11$; $p=0.001$), Seizures ($\chi2=58.73$; $p<0.001$) and AED($\chi2=58.73$; $p<0.001$) were found significant ($\chi2=6.06$; $p=0.048$) with complaints whereas duration of AED and no of AED were all found non significant [Figure 1].

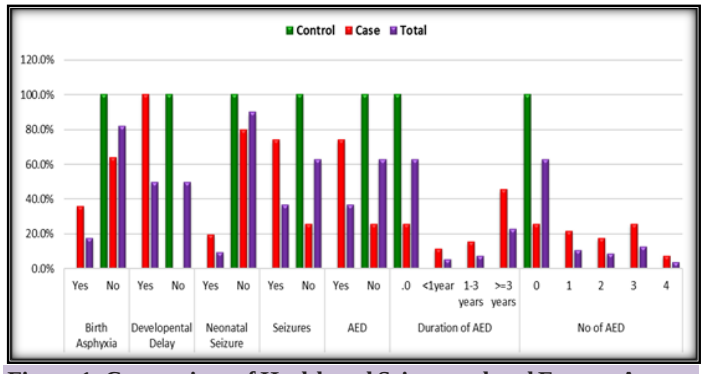

Figure 1: Comparison of Health and Seizure-related Factors Among Control, Case, and Total Groups

The prevalence of decreased vitamin D in patients with CP was 67%. Whereas in our control population the prevalence of decreased vitamin D was 42%. The prevalence of vitamin D deficiency in study group was 21% and insufficiency was 46%. In the control group, vitamin D deficiency amounted to 4%, while insufficiency amounted to 38%. In both populations, the prevalence of insufficiency was more frequent than deficiency. There is a significant association between low vitamin D in CP children when compared to normal population. The prevalence of decreased vitamin D in cases was 67% which was statistically significant, while in the control group, decreased vitamin D levels amounted to 42%.

The results based on Distribution of Factors of Vitamin D deficiency and comparison between Control & Cases illustrated that Dental Changes($\chi2=35.14$;$p<0.001$), Fractures ($\chi2=4.17$; $p=0.041$) and Microcephaly ($\chi2=75.44$; $p<0.001$) were found significant with Vitamin D deficiency.

Further the factor Sunlight Exposure ($\chi2=51.52$; $p<0.001$) was significantly associated with Vitamin D deficiency.

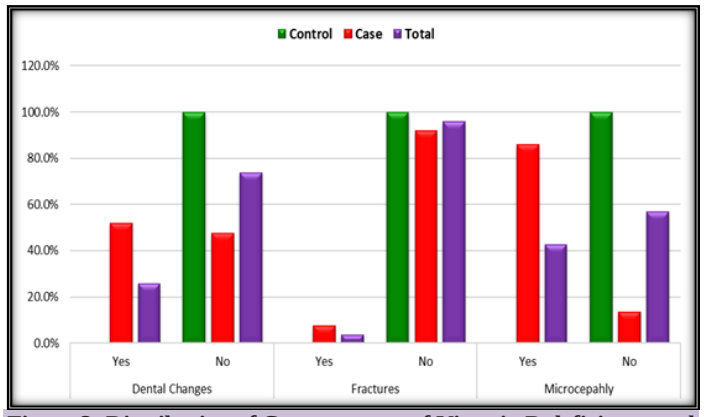

Figure 2: Distribution of Consequences of Vitamin D deficiency and comparison between Control & Cases

Table 1: Distribution of Neonatal Attributes and comparison between Control & Cases

Neonatal Attributes		Control		Case		Total		Significance	
		No.	%	No.	%	No.	%	chi sq	p-value
Sex	Male	26	52.0%	26	52.0%	52	52.0%	0.00	1.000
	Female	24	48.0%	24	48.0%	48	48.0%		
Mode of Delivery	Normal	25	50.0%	30	60.0%	55	55.0%	6.06	0.048
	Elective LSCS	10	20.0%	2	4.0%	12	12.0%		
	Emergency LSCS	15	30.0%	18	36.0%	33	33.0%		
Gestation	Preterm	5	10.0%	8	16.0%	13	13.0%	0.80	0.372
	Term	45	90.0%	42	84.0%	87	87.0%		
	Post term	0	0.0%	0	0.0%	0	0.0%		
Birth Weight	< 2.5 kg	3	6.0%	8	16.0%	11	11.0%	2.55	0.110
	>= 2.5 kg	47	94.0%	42	84.0%	89	89.0%		

Table 2: Serum Vitamin D level and it's comparison between Control & Cases

Neonatal Attributes		Control		Case		Total		Significance	
		No.	%	No.	%	No.	%	chi sq	p-value
Vitamin D	<12	2	4.0%	19	38.0%	21	21.0%	34.09	<0.001
	12 -20	19	38.0%	27	54.0%	46	46.0%		
	> 20	29	58.0%	4	8.0%	33	33.0%		

Discussion

Vitamin D deficiency is a common association in children with Cerebral Palsy due to known reasons like poor sunlight exposure, non-ambulatory nature, anticonvulsant use and feeding difficulties. Though the association is existent and well described in literature, the epidemiological data available regarding the same is less. This gives the reason to implore the study in order to highlight the proportion of children with cerebral palsy who have Vitamin D deficiency and to compare them with normal controls to find statistical significance and also to identify the possible risk factors causing it to enable early identification, periodic monitoring and supplementation with calcium and vitamin D to prevent the development of fractures and deformities.

This present study correlates vitamin D status in children with cerebral palsy in relation to the nutritional status, anticonvulsant use, feeding difficulty, poor sunlight exposure, and type of CP and functional grade of CP which have been implicated as possible causes for vitamin D deficiency in these children.

Vitamin D and CP: It is a widespread misconception that rickets and vitamin D deficiency are uncommon in India, a tropical nation, due to the country's extensive exposure to sunshine. However, now there is proof that the fore mentioned claim is false, which can be justified by the latest studies. All age groups, including newborns, toddlers, schoolchildren, pregnant women, and adult males and females living in India's rural and urban areas, have been well-documented to suffer from vitamin D deficiency.

Studies from North and South India 14-16 claim that between 75 and 85% of the populations examined suffer from vitamin D deficiency or insufficiency to varied degrees. The fact that dietary vitamin D consumption, nutrition, and quantity of solar exposure have a bigger influence on the vitamin D levels in the population is another significant finding from these research. One study revealed a significant effect of dietary calcium supplementation in the study group.

As a consequence of several variables, such as hormonal imbalance, diet and degree of disability, that influence the beginning of puberty, skeletal growth in children with CP may be delayed. Growth retardation and poor skeletal development

may be brought on by feeding issues brought on by trouble swallowing, difficulty controlling the lips and tongue, dental issues malabsorption syndromes and hepatic, renal, and endocrine abnormalities. The correct growth and mineralization of the skeleton depend on adequate vitamin D levels. According to the current study's findings, children with intellectual delay, dental issues, and growth retardation had considerably lower 25(OH)D levels than children who did not have any of these issues.[17]

It is difficult to compare the findings of published studies by different authors and has hampered comparisons between diverse groups and populations since different writers use different cut off values for vitamin D levels for insufficiency and deficiency. Additionally, there are differences in the latitude, solar zenith angle, ultraviolet radiation, amount of cloud cover, duration of exposure to sunlight, and body surface area exposed to sunlight in various regions of the world and within our nation, making comparisons between the various groups impossible. Prevalence in the study group was 21 %, whereas they were 4% in the control group. This statistically significant increase in the prevalence of low vitamin D levels in children with cerebral palsy is not an accident; rather, it is caused by several factors, including poor nutrition caused by feeding and swallowing difficulties, insufficient consumption of calcium-rich foods, inadequate exposure to sunlight, and an increased burden from anti-epileptic medications. None of the controls had any clinical evidence of vitamin D insufficiency, but few children had altered levels of blood calcium and alkaline phosphatase.

Hendersen et al,[18] reported a prevalence of vitamin D deficit in CP children of 19%, which is almost equal to the current study's prevalence of 21%. Similar results were observed by Pinar et al,[17] when they evaluated the vitamin D levels in CP patients. They discovered that 33.6% of them fell into the insufficiency group and that 26.4% had deficient levels. The statistical backup of both, Pinar et al and present study is on similar lines. Seth et al,[19] conducted a study on effect of impaired ambulation and anti-epileptic drug intake on vitamin D status of children with cerebral palsy in Delhi. The present research's location is close to Delhi, with the same daylight, comparable sunlight exposure and cloud coverage, so the vitamin D levels and result analysis will be similar with the study population. In the forementioned research, the mean vitamin D levels of CP patients were 35.6 nmol/L, compared to 60 nmol/L for the control group. In this study, mean Vitamin D levels of 33.82 nmol/L among the cases and in our control population, mean vitamin D value of 55.15 nmol/L which is almost comparable. In the current study, the prevalence of deficiency and insufficiency was 4% and 38%, respectively, in the control group. While previous studies have utilised a higher cut off of <20 ng/ml to designate deficient conditions, this study's cut off value for deficiency is <10 ng/ml, comparable to 12 ng/ml. In this research, the case group's mean vitamin D levels were 13.53 ng/ml, compared to 23.32 ng/ml in the control group. The number of children in the vitamin D deficient group in the aforementioned studies rises when either 15 ng/ml or 20 ng/ml are used as cutoff values in these reference studies. 12ng/ml is the commonly used cutoff point for vitamin D levels. 20 However, given that the parathormone level begins to rise at these levels of 25 OH vitamin D and is utilised as a cut off in the US and other nations, some studies have suggested raising the threshold off to 15 or 20 ng/ml. As of now, there has been no suggestion that India's cutoff threshold for vitamin D be changed. So the study was conducted with cut off value for 25 OH vitamin D deficiency as 12 ng/ml which is recommended by IOM.

Usage of Antiepileptics & Seizures vs Vitamin D

In a population-based research, children with CP made up 38% of the population. However, in this study, 74% of the children experienced seizures. Due to our hospital's status as a tertiary care facility and the greater referral rate for refractory seizures, there is a higher prevalence of seizure disorder in patients with cerebral palsy in this research. The majority of the 37 participants (74%) in this research were taking anticonvulsants, with valproate making about 60% of the total. Levetiracetam was prescribed to 42% of children and phenytoin to 46% of youngsters. Though there was a strong correlation between vitamin D status and certain AEDs, most patients were receiving polytherapy. Thus, no inference can be made for which a specific AED was the cause of vitamin D insufficiency/deficiency.

Duration of AED vs. Vitamin D

It has been discovered that the length of AED use has a statistically significant impact on vitamin D levels. The change is greater when the period of consumption is longer. This has also been consistent with past research that links prolonged anticonvulsant use with changes to low levels and a reduction in BMD. Numerous studies have linked the use of anticonvulsants with altered calcium, vitamin D and bone metabolism. Decreased blood calcium, phosphorus, and 25- hydroxycholecalciferol (calcidiol), as well as enhanced alkaline phosphatase (AP), are examples of biochemical abnormalities. At the same time, other researchers have not identified a connection between anticonvulsant usage and any of these parameters. In the current investigation, low blood phosphorous levels have been strongly linked to vitamin D insufficiency. Though deficient, vitamin D illness is likely in a compensated stage because of elevated parathormone levels; otherwise, doing sonic calcium assays would likely better show low calcium levels.[20]

Fractures & Dental changes vs Vitamin D

In children with neurological impairments, osteoporosis is common.[21] In addition to the detrimental effects of immobility, lack of weight-bearing, and impact of anti-convulsant usage on bone health, inadequate availability of essential nutrients for bone formation may also result in decreased muscular strength. Only 4 (8%) of the children in the research experienced fractures, and the correlation between this with vitamin D levels was not statistically significant. Three of the four children who suffered fractures were quadriplegics, which is often how fractures are connected with quadriplegics. In residential settings, children and adolescents with quadriplegic cerebral palsy had a higher incidence of long-bone fractures. So, a conclusion can be made that the greatest factor for the development of fractures in the group under study was vitamin D insufficiency. It can be concluded that the occurrence of fractures was a result of vitamin D deficiency in the population studied.[3]

On the other hand, when compared to vitamin D levels, dental alterations were seen in 52% of people, which was statistically significant.

Limitations and recommendations

The research would have been more comprehensive if the levels of vitamin D and parathormones had been compared, but this was not possible owing to financial limitations. To make step-by-step suggestions, a bigger prospective case-control research will provide considerably more information. This study has not measured bone mineral density in the participants, was another drawback since, in the absence of any literature on the issue in Indian children, this would have shed light on the bone mineral density status of children with cerebral palsy. The inability to properly execute the test on these individuals, the lack of reference standards for Indian youngsters to compare the findings to and the difficulty in matching were the reasons bone mineral density tests were not performed. Because the test was expensive to do, controls like healthy volunteers could not be compared to children with CP. Given that vitamin D insufficiency is avoidable, a proactive strategy is needed to identify these possible risk factors, detect them, and treat them as necessary. To find malnourished children, routine nutritional evaluation is required. Introducing a gastrostomy tube should be taken into account early on to promote proper feeding. It's important to emphasise that parents need to be educated about the importance of getting enough sunshine. In individuals at risk, calcium and vitamin D supplements should be taken into consideration. Further research is necessary to reach a consensus about preventive calcium and vitamin D supplements, their dose, and the length of therapy in high-risk categories like cerebral palsy since vitamin D deficiency is believed to be common in otherwise healthy patients.

Conclusion

The presence of feeding difficulties, poor sunlight exposure, poor nutritional status, and the use of antiepileptic drugs, type of CP and the functional grade of CP had statistically significant association with Vitamin D deficiency in these children. Periodic monitoring, early identification and appropriate calcium and vitamin D supplements may prevent complications like fractures, etc.

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Conflicts of interest

There are no conflicts of interest.

References

1. Richards CL, Malouin F. Cerebral palsy. Handbook of Clinical Neurology. 2013;:183–95.
2. Chen R, Sjölander A, Johansson S, Lu D, Razaz N, Tedroff K, et al. Impact of gestational age on risk of Cerebral Palsy: Unravelling the role of neonatal morbidity. International Journal of Epidemiology. 2021;50(6):1852–63.
3. Mushta SM, King C, Goldsmith S, Smithers-Sheedy H, Badahdah A-M, Rashid H, et al. Epidemiology of Cerebral Palsy Among Children and Adolescents in Arabic-speaking countries: A systematic review and meta-analysis. Brain Sciences. 2022;12(7):859.
4. Vincer MJ, Allen AC, Allen VM, Baskett TF, O'Connell CM. Trends in the prevalence of cerebral palsy among very preterm infants (<31 weeks' gestational age). Paediatrics & Child Health. 2014;19(4):185–9.
5. Serenius F, Ewald U, Farooqi A, Fellman V, Hafström M, Hellgren K, et al. Neurodevelopmental outcomes among extremely preterm infants 6.5 years after active perinatal care in Sweden. JAMA Pediatrics. 2016Oct1;170(10):954.
6. Tseng S-H, Lee J-Y, Chou Y-L, Sheu M-L, Lee Y-W. Association between socioeconomic status and cerebral palsy. PLOS ONE. 2018;13(1).
7. Agarwal A, Verma I. Cerebral palsy in children: An overview. Journal of Clinical Orthopaedics and Trauma. 2012;3(2):77–81.
8. Patel DR, Neelakantan M, Pandher K, Merrick J. Cerebral palsy in children: A clinical overview. Translational Pediatrics. 2020;9(S1).
9. Sadowska M, Sarecka-Hujar B, Kopyta I. cerebral palsy: Current opinions on definition, epidemiology, risk factors, classification and treatment options. Neuropsychiatric Disease and Treatment. 2020;Volume 16:1505–18.
10. Toopchizadeh, Vahideh et al. "Prevalence of Vitamin D Deficiency and Associated Risk Factors in Cerebral Palsy A study in North-West of Iran." Iranian journal of child neurology vol. 12,2 (2018): 25-32.
11. Ajami S, Maghsoudlorad AA. The Role of Information Systems to Manage Cerebral Palsy. Iran J Child Neurol.Spring. 2016;10(2):1–9
12. Alenazi KA. Vitamin D deficiency in children with cerebral palsy: A narrative review of epidemiology, contributing factors, clinical consequences and interventions. Saudi Journal of Biological Sciences. 2022;29(4):2007–13.
13. Chutterpaul P, Paruk F, Cassim B. Prevalence of vitamin D deficiency in older South Africans with and without hip fractures and the effects of age, body weight, ethnicity and functional status. Journal of Endocrinology, Metabolism and Diabetes of South Africa. 2018;24(1):10–5.
14. Park MJ, Yoo YJ, Chung CY, Hwang J-M. Ocular findings in patients with spastic type cerebral palsy. BMC Ophthalmology. 2016;16(1).
15. Pavone, P., Gulizia, C., Le Pira, A., Greco, F., Parisi, P., Di Cara, G., Falsaperla, R., Lubrano, R., Minardi, C., Spalice, A., & Ruggieri, M. (2020). Cerebral Palsy and Epilepsy in Children: Clinical Perspectives on a Common Comorbidity. Children (Basel, Switzerland), 8(1), 16.
16. Dziadosz M, Evans MI. Re-thinking elective single embryo transfer: Increased risk of monochorionic twinning - A systematic review. Fetal Diagnosis and Therapy. 2017;42(2):81–91.
17. Akpınar P. Vitamin D status of children with cerebral palsy (should vitamin D levels be checked in children with cerebral palsy?). Northern Clinics of Istanbul. 2018;
18. Metz C, Jaster M, Walch E, Sarpong-Bengelsdorf A, Kaindl AM, Schneider J. Clinical phenotype of cerebral palsy depends on the cause: Is it really cerebral palsy? A retrospective study. Journal of Child Neurology. 2021;37(2):112–8.
19. Dreher T, Thomason P, Švehlík M, Döderlein L, Wolf SI, Putz C, et al. Long-term development of gait after multilevel surgery in children with cerebral palsy: A multicentre cohort study. Developmental Medicine & Child Neurology. 2017;60(1):88–93.
20. Rathi N, Rathi A . Vitamin D and child health in the 21st century. Indian Pediatrics 2011;48:619-625
21. Ko A, Kong J, Samadov F, Mukhamedov A, Kim YM, Lee Y-J, et al. Bone Health in pediatric patients with neurological disorders. Annals of Pediatric Endocrinology & Metabolism. 2020;25(1):15–23.