

Mini Review

Nutritional interventions in children with cerebral palsy: a mini review

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Abstract

The aim of the present study was to review the current knowledge concerning the nutritional status of children with cerebral palsy. The aim was also to summarize the key factors which are responsible for the high prevalence of malnutrition. Significant reductions in anthropometric parameters were found in children with cerebral palsy in comparison to children of normal development. This deviation from typical growth, can be attributed to several factors, such as: inadequate food intake, deficiency of important trace elements (calcium, phosphorus, magnesium, iron) and vitamins (vitamin D, folate, vitamin 12) and reduced dietary density (which depends on dietary factors and mobility). Children with cerebral palsy, manifest nutritional problems and nutritional deficiency, which mainly is due to the inherent problems these children have to acquire dietary factors and their also inherent problematic in their mobility. Thus the present work attempts to provide a short review of the nutritional issues in children with cerebral palsy and the probable nutritional therapeutic interventions.

Keywords: Nutrition, Cerebral palsy, Nutritional interventions, Childhood malnutrition

Introduction

Cerebral Palsy (CP) is a condition, which can be defined as "a heterogeneous group of nonprogressive motor disorders caused by chronic brain injuries that originate in the prenatal period, perinatal period, or first few years of life. The four major subtypes are spastic, athetoid, ataxic, and mixed cerebral palsy, with spastic forms being the most common. The motor disorder may range from difficulties with fine motor control to severe spasticity (such as muscle spasticity) in all limbs. Spastic diplegia (Little disease) is the most common subtype, and is characterized by spasticity that is more prominent in the legs than in the arms. Pathologically, this condition may be associated with leukomalacia"¹.

CP comprises of a set of conditions caused by interference to the development of the brain, conventionally before birth, which has an impact on a person's movement balance and posture. There are cases in which some genetic defect may exist resulting in "miss-wiring" in the course of the development of the neuronal system. Cerebral palsy is typically diagnosed during infancy or preschool whereas the effect on physical ability varies notably²⁻⁵. Cerebral palsy can result in impaired movement accompanied by exaggerated reflexes, floppiness or rigidity of the limbs and trunk, abnormal posture, involuntary movements and

unsteadiness of walking^{6,7}. Mental disabilities have also been detected, although some people show no or little intellectual dysfunction. Epilepsy, blindness or deafness may also occur^{7,8}.

Etiology

The etiology of cerebral palsy is multifactorial. In a large proportion of patients (30%) there is no specific risk factor or recognized etiology. Risk factors for CP are divided into three: prenatal, perinatal and postnatal factors^{8,9}. According to epidemiological studies of already patients with cerebral palsy, 70% to 80% of cases are due to prenatal causes, and perinatal asphyxia represents less than 10% of cases. Of the above, the main risk factors

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are premature life, multiple pregnancies and maternal infections such as chorioammonitis^{9,10}.

Clinical presentation of children with cerebral palsy

Conforming to the patient's clinical presentation, cerebral palsy has been classified according to the nature of the kinetic disorder (spasticity, ataxia, dystonia and athetosis) and the anatomical or topographical distribution of motor abnormalities^{7,8}. The most common clinical picture among premature babies is spasticity, which is dependent on the speed of muscle tone¹¹⁻¹³. A minority of cases, reported dyskinetic cerebral palsy, the predominant abnormality is either dystonia or choreo-athetosis.

Dystonia is expressed in hypertonia and decreased activity, choreoathetosis, to irregular, spasticity, involuntary movements of the limbs or facial muscles. With ataxic cerebral palsy there is a loss of orderly muscular coordination, so that movements are performed with abnormal force, rhythm, and accuracy¹¹⁻¹³. In addition, spasticity in cerebral palsy is classified into spastic diplegia (bilateral spasticity with leg-to-arm legs), spastic hemiplegia (unilateral spasticity), or quadriplegia (bilateral spasticity with an arm equal to or greater than the leg). In a large percentage of patients with cerebral palsy, there are secondary concomitant diseases such as musculoskeletal problems, epilepsy and sensory, perception, knowledge, communication and behavior disorders¹⁴.

The role of nutrition

Malnutrition and poor growth have been surveyed, in several studies of cerebral palsy because of its effects on the psychological, physiological eustasis, motor function, social participation, and survival. In an effort to understand the causes of poor growth, a range of interventions have been implemented. Poor development is mainly caused by malnutrition. Maintaining balanced and rich diet has helped to improve clinical management and thus survival in the last 20 years, according to scientific evidence. Furthermore, the acknowledgement of the effect neurological, endocrinological and environmental factors have on CP, has set new standards in the treatment of suffering children...

Difficulty in feeding and nutrient absorption in children with cerebral palsy

Data collected has shown that swallowing and chewing difficulties^{15,16}, inability to feed and long meal times are some of the reasons that cause such a great percentage of children suffering from CP to have slowed down or degraded growth, as they contribute to malnutrition^{17,18}. Although there have been many reports of unsatisfactory uptake of minerals and vitamins (micronutrients), modern biochemical methods have only scarcely measured their state¹⁹⁻²¹. Low energy levels that have often been observed in children with neurological disability may be the cause of insufficient consumption of micronutrients^{15,20}.

Metabolic deficiencies disrupt metabolism in such a way that a sufficient intake of micronutrients is essential. According to a micronutrient-status study that monitored children with cerebral palsy, it appeared that cerebral palsy symptoms due to micronutrient inadequacy are not easily distinguished from the typical CP clinical presentation²².

Micronutrient and vitamins status in children with cerebral palsy

A number of studies have been carried out over the last few years to investigate the micronutrient status in a group of children with CP. In a recent survey thirty six children with CP were screened during the procedure, aged 1.5–17 years, who completed a 4-day food diary, underwent anthropometric measurements and delivered blood for analysis of micronutrient concentrations. The findings of the survey indicate that, even among those who were receiving nutritional supplements, inadequate intake of iron, niacin, folate, vitamin E, calcium and vitamin D was recurrent. Clinical tests also revealed low serum concentration of folate, tocopherol, ferritin and pyridoxal-5-phosphate. Severely disabled children received nutrition supplements more systematically than those with less severe disability (71% vs. 16%, $p=0.01$). Tube feeding and use of nutrition supplements had as an outcome higher concentrations of micronutrients in serum and blood.

All the above data indicate that it was common among the children of this heterogenic group to demonstrate low intake of micronutrients as well as low micronutrient concentrations. Moreover, children with neurological disabilities ought to have their nutritional status evaluated in order to make sure they intake adequate amount of micronutrients²³. Lack of leaflet can cause chromosome breakage in human genes and has been related to cognitive impairment in children. Lower than expected folate concentrations were concurrent with low or marginal folate intake in several children. Also, many specimens displayed low quantities of vitamin E and serum. What ought to be diagnosed and treated is iron inadequacy as it may affect learning as well as typical hemoglobin levels²³. Previous reports have shown that children with severe CP have a high risk of fracture²⁴. Thus, a significant preventive factor is the adequate intake of magnesium, vitamin D and calcium, which should consist of a minimum regulatory nutrient intake. Unsatisfactory supply of critical nutrients for bone formation deteriorates the negative effect of immobility, weight loss and the use of anticonvulsants on bone health and may also be responsible for decreased muscle strength²⁵. The ideal levels of vitamin D are linked, to a large extend, to adequate sun exposure.

Further study is needed though, as far as food intake is concerned, because the most trusted method of calculating food intake in an individual level is recorded by weighing food²⁶, which is regarded as untrustworthy by caretakers of a group of children with spastic quadriplegic cerebral palsy, in spite of careful instructions for preserving food²⁷. The

results, on the other hand, show that quite a few of these micronutrients may be insufficient in this group of patients, even though the nutritional deficiency is differentiated from the general harm of children with neurological disease. The inadequate consumption of micronutrients and the biochemical deficiency of numerous micronutrients have frequently been encountered in studies of children with CP. Higher micronutrient consumption, and thus higher concentrations of micronutrients in blood and serum is what the use of dietary supplements aims at. The evaluation of the nutritional status of children with disabilities is essential, together with laboratory and dietary assessment. Nutrition advice should be personalized so that the use of nutritional supplements can assist development and diminish the hazard of inadequacy of micronutrients in children with serious drawbacks.

Vitamin D and Bone mineral density status in children with cerebral palsy

Calcium and vitamin D deficiencies have also been investigated in several studies, where it has been shown that children with CP were prone to vitamin D deficiency²⁸⁻³¹ as well as to calcium abnormal levels^{32,33}. It is commonly accepted that immobilization predisposes to bone absorption, which can reduce bone density increasing the risk of anomalies. Therefore, children that are mostly in danger of encountering such problems are those who suffer from physical handicaps that make them immobile, for instance those with severe cerebral palsy.

Vitamin D is a necessary vitamin for bone metabolism, which has the potential to be synthesized on the skin.

Nowadays, in order to avoid vitamin D deficiency, milk is supplemented with vitamin D, but milk products such as cheese, yoghurt and ice cream are not generally supplemented with vitamin D. There are few foods that contain naturally significant amounts of vitamin D, including fatty fish and fish oils²⁴. It is known that lack of vitamin D in children causes rickets. However, this is considered to be a regional effect since most reports concern developing countries^{31,34}. Further on, several studies have highlighted the fact that vitamin D deficiency along with cerebral palsy, is a probable cause for spontaneous fractures in children³⁵⁻³⁷. There is not an exact explanation for this phenomenon, since it is multifactorial and include many other causative factors. Some of these reasons, include the limiting the exposure of children with CP to sunlight, feeding difficulties resulting in malabsorption of nutrients, the long-term of anticonvulsants and antiepileptics and others.

Reduced bone density, as evidenced by DEXA bone densitometry and the tendency towards low-grade fractures and extensive bone desalting, are common in children with cerebral palsy³⁸. Long bone fractures are common among this group of children, leading to increased and prolonged hospitalization and deteriorating the quality of life of this already vulnerable group. A potential therapeutic opportunity

can be offered through the use of bisphosphonates³⁹. Recent studies have highlighted the therapeutic potential of bisphosphonates in children with cerebral palsy, where it has been shown that children treated with bisphosphonates had significant lower fracture incidents^{24,40,41}. In other studies, a group of children with severe cerebral palsy were examined for the prevalence of osteopenia and determination of its relation to irregularities in the vitamin D or parathyroid hormone status. Those studies highlighted that there was a significant difference in osteopenia levels, where children receiving medical and nutritional supplementation manifested increased levels of BMD^{19,20,24}.

As aforementioned, children with cerebral palsy (CP) are at higher risk of low bone mineral density (BMD). The lack of mobility of children with CP is another risk factor for low bone density. An interesting study, conducted in ambulatory and non-ambulatory children with cerebral palsy, evaluated the margins of Z-bone femoral and lumbar spine (BMD) clearance. The BMD z-score was found to be higher in ambulatory children than in non-ambulatory individuals.

In a recent study the distal femur and lumbar spine bone mineral density (BMD) as well as Z-scores in children with cerebral palsy were evaluated. BMD z-score was found to be higher in ambulatory children than in non-ambulatory. To a certain degree, unexpectedly, even among ambulatory children, those with better walking abilities had higher BMD z-score than those with more impaired walking ability. From this we conclude that the main prognosticator of low BMD Z-scores in the distal femur was the inability to walk, but the outcomes suggest that the degree of the neuromotor impairment may possibly be an important predictor. Vitamin D status did not seem to have any connection to BMD z-scores^{24,42-44}.

From the aforementioned study it was concluded that a considerable number of children suffering from CP had low BMD for age in the peripheral femur and that mostly children who could not walk had very low z-BMD scores. Quite unexpectedly, it was found that among children who were able to walk, those with GMFCS level II had considerably lower Z-scores than GMFCS I children. Furthermore, among children with hemiplegia, BMD Z scores were found to be lower in the infected than the unaffected end^{24,43,45-47}.

Conclusions

Low BMD in children with CP is the cause of brittle bones and bone fractures, and thus deteriorates their quality of life⁴⁴. It is admittedly almost impossible for optimal maximum bone mass to be obtained, due to the fact that risk aspects for low BMD appear from a very early age in children with CP. Therefore, what is of crucial importance is to recognize children at risk as early as possible, so as to improve bone growth. Proper nutrition, emphasizing on calcium and vitamin D intake and premature mobilization can improve bone acquisition, although normal bone growth is unlikely to be expected⁴⁸. The aforementioned outcomes

assist the weight of the activity level as well as the degree of neuromuscular injury which play an essential role in bone modeling in this population.

It is proven that if a child with CP has reduced mobility they are at increased risk of developing low bone density or osteoporosis/osteopenia. Although, there is not a commonplace for CP patients' diet it is agreed that an adequate intake of calcium and Vitamin D is imperative. Furthermore, food and diet is a highly regional and customs-related aspect, which however needs to cover basic nutritional needs. For example, calcium-rich foods such as milk, cheese, yogurts, milk puddings, dark green leafy vegetables, tinned fish and sesame seeds/paste are necessary. Further on, Vitamin D is imperative, which is found in egg yolk, liver, oily fish, fortified milk or spreads, and through 15-20 mins exposure to sunlight particularly between the months of March to September. In case that these foods are disliked, non-digestible or out of a custom-related diet then a trained dietitian should be involved in order to supply the necessary nutritional factors. In addition, a child with CP should have their bone health monitored by blood tests and bone density scans. This is especially important if anti-epilepsy medication is used to control seizures. Consider some weight-bearing exercise, in consultation with a physiotherapist.

In relation to the aforementioned studies, there is a common consensus for the exact nutrition of children with CP. However, it is generally accepted that the energy requirements, in children with CP, are averagely lower than that of healthy children of the same age, varies highly and does not seem to be affected by any body composition factor. It is therefore quite difficult to evaluate the energy balance using the currently available equations. As a result, the recommendation at present is either to measure Resting Energy Expenditure (REE) by indirect calorimetry, even if this multifaceted technology might not be accessible at all centers or to evaluate the energy necessities using the existing available equations and to control energy consumption according to weight modification. Last but not least, the conducted studies have revealed that the low energy expenditure discovered in malnourished children with CP along with low levels of trace minerals and vitamins is partly a result of an adaptation to chronic low energy consumption and that REE of CP babies can return to normal with sufficient nutritional intake.

References

1. Badawi N, Watson L, Petterson B, Blair E, Slee J, Haan E, et al. What constitutes cerebral palsy? *Developmental medicine and child neurology* 1998;40(8):520-7.
2. Boxum AG, Dijkstra LJ, la Bastide-van Gemert S, Hamer EG, Hielkema T, Reinders-Messelink HA, et al. Development of postural control in infancy in cerebral palsy and cystic periventricular leukomalacia. *Research in developmental disabilities* 2018;78:66-77.
3. Hamer EG, La Bastide-Van Gemert S, Boxum AG, Dijkstra LJ, Hielkema T, Jeroen Vermeulen R, et al. The tonic response to the infant knee jerk as an early sign of cerebral palsy. *Early human development* 2018;119:38-44.
4. van Balen LC, Boxum AG, Dijkstra LJ, Hamer EG, Hielkema T, Reinders-Messelink HA, et al. Are postural adjustments during reaching related to walking development in typically developing infants and infants at risk of cerebral palsy? *Infant behavior & development* 2018;50:107-15.
5. Novak I, Morgan C, Adde L, Blackman J, Boyd RN, Brunstrom-Hernandez J, et al. Early, Accurate Diagnosis and Early Intervention in Cerebral Palsy: Advances in Diagnosis and Treatment. *JAMA pediatrics* 2017;171(9):897-907.
6. Graham HK, Rosenbaum P, Paneth N, Dan B, Lin JP, Damiano DL, et al. Cerebral palsy. *Nature reviews Disease primers*. 2016;2:15082.
7. Colver A, Fairhurst C, Pharoah PO. Cerebral palsy. *Lancet (London, England)* 2014;383(9924):1240-9.
8. Kuban KC, Leviton A. Cerebral palsy. *The New England journal of medicine* 1994;330(3):188-95.
9. Jacobsson B, Hagberg G. Antenatal risk factors for cerebral palsy. *Best practice & research Clinical obstetrics & gynaecology* 2004;18(3):425-36.
10. Economic costs associated with mental retardation, cerebral palsy, hearing loss, and vision impairment - United States, 2003. *MMWR Morbidity and mortality weekly report* 2004;53(3):57-9.
11. Chabrier S, Husson B, Dinomais M, Landrieu P, Nguyen The Tich S. New insights (and new interrogations) in perinatal arterial ischemic stroke. *Thrombosis research* 2011;127(1):13-22.
12. O'Shea TM. Cerebral palsy in very preterm infants: new epidemiological insights. *Mental retardation and developmental disabilities research reviews* 2002;8(3):135-45.
13. Van Steenwinckel J, Schang AL, Sigaut S, Chhor V, Degos V, Hagberg H, et al. Brain damage of the preterm infant: new insights into the role of inflammation. *Biochemical Society transactions* 2014;42(2):557-63.
14. Cooley WC. Providing a primary care medical home for children and youth with cerebral palsy. *Pediatrics* 2004;114(4):1106-13.
15. Krick J, Murphy-Miller P, Zeger S, Wright E. Pattern of growth in children with cerebral palsy. *Journal of the American Dietetic Association* 1996;96(7):680-5.
16. Vik T, Skrove MS, Dollner H, Helland G. [Feeding problems and growth disorders among children with cerebral palsy in south and north Trondelag]. *Tidsskrift for den Norske laegeforening : tidsskrift for praktisk medicin, ny raekke* 2001;121(13):1570-4.
17. Sullivan PB, Lambert B, Rose M, Ford-Adams M, Johnson A, Griffiths P. Prevalence and severity of feeding and nutritional problems in children with neurological impairment: Oxford Feeding Study. *Developmental medicine and child neurology* 2000;42(10):674-80.
18. Thommessen M, Kase BF, Riis G, Heiberg A. The impact of feeding problems on growth and energy intake in children with cerebral palsy. *European journal of clinical nutrition* 1991;45(10):479-87.
19. Hals J, Ek J, Svalastog AG, Nilsen H. Studies on nutrition in severely neurologically disabled children in an institution. *Acta paediatrica (Oslo, Norway: 1992)* 1996;85(12):1469-75.
20. Jones M, Campbell KA, Duggan C, Young G, Bousvaros A, Higgins L, et al. Multiple micronutrient deficiencies in a child fed an elemental formula. *Journal of pediatric gastroenterology and nutrition* 2001;33(5):602-5.
21. Sanchez-Lastres J, Eiris-Punal J, Otero-Cepeda JL, Pavon-Belinchon P, Castro-Gago M. Nutritional status of mentally retarded children in northwest Spain: II. Biochemical indicators. *Acta paediatrica (Oslo, Norway: 1992)* 2003;92(8):928-34.
22. Azcue MP, Zello GA, Levy LD, Pencharz PB. Energy expenditure and body composition in children with spastic quadriplegic cerebral palsy. *The Journal of pediatrics* 1996;129(6):870-6.
23. Lozoff B, Jimenez E, Wolf AW. Long-term developmental outcome

- of infants with iron deficiency. *The New England journal of medicine* 1991;325(10):687-94.
24. Henderson RC, Lark RK, Gurka MJ, Worley G, Fung EB, Conaway M, et al. Bone density and metabolism in children and adolescents with moderate to severe cerebral palsy. *Pediatrics* 2002;110(1 Pt 1):e5.
 25. Duncan B, Barton LL, Lloyd J, Marks-Katz M. Dietary considerations in osteopenia in tube-fed nonambulatory children with cerebral palsy. *Clinical pediatrics* 1999;38(3):133-7.
 26. Bingham SA. The dietary assessment of individuals; methods, accuracy, new techniques and recommendations 1987.
 27. Stallings VA, Zemel BS, Davies JC, Cronk CE, Charney EB. Energy expenditure of children and adolescents with severe disabilities: a cerebral palsy model. *The American journal of clinical nutrition* 1996;64(4):627-34.
 28. Lee JJ, Lyne ED, Kleerekoper M, Logan MS, Belfi RA. Disorders of bone metabolism in severely handicapped children and young adults. *Clinical orthopaedics and related research*. 1989(245):297-302.
 29. Akpinar 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;5(4):341-7.
 30. 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. *Paediatrics and international child health* 2017;37(3):193-8.
 31. Toopchizadeh V, Barzegar M, Masoumi S, Jahanjoo F. Prevalence of Vitamin D Deficiency and Associated Risk Factors in Cerebral Palsy A study in North-West of Iran. *Iranian journal of child neurology* 2018;12(2):25-32.
 32. Sangermano M, D'Aniello R, Massa G, Albano R, Pisano P, Budetta M, et al. Nutritional problems in children with neuromotor disabilities: an Italian case series. *Italian journal of pediatrics* 2014;40:61.
 33. Jaramillo C, Johnson A, Singh R, Vasylyeva TL. Metabolic disturbances in patients with cerebral palsy and gastrointestinal disorders. *Clinical nutrition ESPEN* 2016;11:e67-e9.
 34. Haider N, Nagi AG, Khan KM. Frequency of nutritional rickets in children admitted with severe pneumonia. *JPMA The Journal of the Pakistan Medical Association* 2010;60(9):729-32.
 35. Wick JY. Spontaneous fracture: multiple causes. *The Consultant pharmacist: the journal of the American Society of Consultant Pharmacists* 2009;24(2):100-2, 5-8, 10-2.
 36. Sheth RD. Bone health in pediatric epilepsy. *Epilepsy & behavior : E&B* 2004;5 Suppl 2:S30-5.
 37. De Smet L. Kienbock disease in cerebral palsy. *Acta orthopaedica Belgica* 2001;67(1):81-3.
 38. Favus MJ. *Primer on the metabolic bone diseases and disorders of mineral metabolism: Rittenhouse Book Distributors; 2006.*
 39. Kim MJ, Kim SN, Lee IS, Chung S, Lee J, Yang Y, et al. Effects of bisphosphonates to treat osteoporosis in children with cerebral palsy: a meta-analysis. *Journal of pediatric endocrinology & metabolism: JPEM* 2015;28(11-12):1343-50.
 40. Plotkin H, Coughlin S, Kreikemeier R, Heldt K, Bruzoni M, Lerner G. Low doses of pamidronate to treat osteopenia in children with severe cerebral palsy: a pilot study. *Developmental medicine and child neurology* 2006;48(9):709-12.
 41. Bachrach SJ, Kecskemethy HH, Harcke HT, Lark RK, Miller F, Henderson RC. Pamidronate treatment and posttreatment bone density in children with spastic quadriplegic cerebral palsy. *Journal of clinical densitometry : the official journal of the International Society for Clinical Densitometry* 2006;9(2):167-74.
 42. Mughal MZ. Fractures in children with cerebral palsy. *Current osteoporosis reports* 2014;12(3):313-8.
 43. Henderson RC, Lin PP, Greene WB. Bone-mineral density in children and adolescents who have spastic cerebral palsy. *The Journal of bone and joint surgery American volume* 1995;77(11):1671-81.
 44. Ondrak KS, Morgan DW. Physical activity, calcium intake and bone health in children and adolescents. *Sports medicine (Auckland, NZ)* 2007;37(7):587-600.
 45. Al Wren T, Lee DC, Kay RM, Dorey FJ, Gilsanz V. Bone density and size in ambulatory children with cerebral palsy. *Developmental medicine and child neurology* 2011;53(2):137-41.
 46. Chen CL, Ke JY, Wang CJ, Wu KP, Wu CY, Wong AM. Factors associated with bone density in different skeletal regions in children with cerebral palsy of various motor severities. *Developmental medicine and child neurology* 2011;53(2):131-6.
 47. Henderson RC, Kairalla J, Abbas A, Stevenson RD. Predicting low bone density in children and young adults with quadriplegic cerebral palsy. *Developmental medicine and child neurology* 2004;46(6):416-9.
 48. Mergler S, Evenhuis HM, Boot AM, De Man SA, Bindels-De Heus KG, Huijbers WA, et al. Epidemiology of low bone mineral density and fractures in children with severe cerebral palsy: a systematic review. *Developmental medicine and child neurology* 2009;51(10):773-8.