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Evidence-Based Review of Bone Strength in Children and Youth With Cerebral Palsy

Michal Cohen, MD, Eli Lahat, MD, Tzvy Bistritzer, MD, Amir Livne, MD, Eli Heyman, MD, and Marianna Rachmiel, MD

Children with cerebral palsy have various risk factors for compromised bone health. Evidence concerning their bone fragility is gathering; however, there is no consensus regarding risk factors, indications for evaluation, follow-up, or treatment. We performed an evidence-based review targeted to address the following questions concerning children with cerebral palsy: Is bone strength impaired and what are the risk factors? Are these children at increased risk for bone fractures? What are the relations between bone mineral density and fracture risk? What methods can be used for bone health assessment? How can bone strength be improved? Currently, the most acceptable method for evaluating bone status in children is dual-energy x-ray absorptiometry. Evidence demonstrates reduced bone mass in children with cerebral palsy; yet, no clear association with fractures. Preventive methods are suggested.

Keywords: cerebral palsy; bone; osteopenia; osteoporosis

Cerebral palsy is the most common form of chronic physical disability in childhood; prevalence is variable and estimated at 2 to 4 per1000.[1,2] This is a diverse group of disorders, etiologically and clinically, with motor involvement being a shared characteristic. The possible involvement of bone health in the spectrum of abnormalities is a novel field of research in this population, and there is accumulating evidence demonstrating children and youth with cerebral palsy suffer low or suboptimal bone strength.

Bone strength depends on bone mass and density and on the structural properties of bone.[3] Bone mass and strength during adulthood depend on the peak bone mass reached and the rate of bone loss, and fracture risk in adults is related to those achieved parameters.[4] Peak bone mass is determined mainly (60%-80%) by genetic factors. The additional 20% to 40% are influenced by hormonal balance and extrinsic factors such as diet, weight, lifestyle, and medications.[4-7] As currently assessed by bone mineral density, peak bone mass is normally reached during the first 2 decades of life, thereafter comes a period of steady state followed by a slow, continuous decline in bone mass. Fulfilling one's potential for peak bone mass cannot be overestimated; however, this might be compromised in chronically ill children with disability, such as youth with cerebral palsy. Their possibly reduced bone health might expose them to early fractures and severely impaired quality of life, as well as long-term bone fragility and decreased adult bone strength. Therefore, identifying such "at risk" patients is crucial for preventive medicine and early management.

Osteopenia and osteoporosis in children, not as in adults, are not easily defined and a more preferable term is "low bone mass"; this is defined as a bone mineral content or areal bone mineral density *z* score that is less than or equal to −2.0, adjusted for age, gender, and body size.[8] The diagnosis of osteoporosis according to the 2007 Official Positions of the International Society for Clinical Densitometry requires the presence of both a clinically significant fracture history and a low bone mineral content or a bone mineral density.[8] Osteoporosis is characterized by reduced bone mass and abnormal microarchitecture demonstrated by bone biopsy, which is rarely performed in children.[3]

Fragility fractures and decreased bone mineral density may be caused by rickets as well as osteoporosis. Rickets results from decreased mineralization of the growing bone[9,10]; possible causes are deficiencies of calcium,

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From Maccabi Healthcare Services, Jerusalem & Hashfela District, Israel (MC); Pediatric Neurology & Rehabilitation Division (EL, AL, EH), and Pediatrics B & Pediatric Endocrinology Unit (TB, MR), Assaf Harofeh Medical Center, Zerifin, Israel; and Department of Pediatrics, Sackler Faculty of Medicine, Tel-Aviv University, Israel (MC, EL, TB, AL, EH, MR).

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Address correspondence to: Michal Cohen, MD, Maccabi Healthcare Services, Jerusalem & Hashfela District, Israel; e-mail: cohenm4@gmail.com.

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Table 1. Type of Evidence Definition

Level	Type of Evidence
Ia	Obtained from meta-analysis of randomized controlled trials
Ib	Obtained from at least 1 randomized controlled trial
IIa	Obtained from at least 1 well-designed controlled study without randomization
IIb	Obtained from at least 1 other type of well designed quasi-experimental study
III	Obtained from well-designed nonexperimental, descriptive study, such as comparative study, correlation studies, or case control studies
IV	Obtained from expert committee reports or opinions and/or clinical experience of respected authorities

vitamin D, or phosphorus and less commonly the existence of mineralization inhibitors. Its diagnosis depends on abnormally low levels of serum minerals and typical radiological parameters. In this report, we will refer to low bone mass that is not caused by rickets. It should be common practice that adequate calcium and vitamin D requirements are the first assessment and consideration in children with cerebral palsy and should be addressed and monitored regularly and frequently. Sufficient daily intake of calcium and vitamin D is of major importance in youth in general and in children with cerebral palsy in particular.

The objective of this report was to perform an evidence-based review of bone health in children with cerebral palsy. We aimed to answer a number of questions:

1. Is bone strength impaired in children with cerebral palsy and if so, what are the risk factors for such impairment?
2. Are children with cerebral palsy at increased risk for bone fractures, what are the risk factors, and is it related to bone mineral density?
3. Which methods should be used for bone-health assessment in children with cerebral palsy?
4. How can bone strength be improved in children with cerebral palsy?

Methods

A systematic literature search of PubMed and Cochrane databases between the years 1995 and 2008 was conducted. Search themes included "cerebral palsy," "children," "bone density," "fracture," "prevention," and "treatment" in various combinations. This was supplemented by a manual search of bibliographies of retrieved articles. We considered only articles published in English. The definition of types of evidence follows that of the Agency for Health Care Policy and Research, as presented in Table 1.[11] Because this is a review article, no institutional review board approval or informed consent procedures were needed.

Discussion

Question no. 1a: Is Bone Strength Impaired in Cerebral Palsy?

Bone mineral density and bone mineral content of children with cerebral palsy are lower than age-matched normal values[12-20] (level III),[21](level IIa). Most authors used dual-energy x-ray absorptiometry to measure bone mineral density. Longitudinal follow-up of these children demonstrated bone mineral density to increase by a median of 2.5% per year. This is not a sufficient increment during childhood and youth, and bone mineral density *z* scores, the standard deviation from age- and gender-specific norms decreased.[14] This highlights the main difference between adult osteoporosis, caused by bone mineral loss, and childhood decreased bone mass when insufficient bone mineralization is taking place.

Tibial peripheral quantitative computed tomography demonstrated lower cortical bone mineral content, area and thickness, polar strength-strain index and periosteal circumference in cerebral palsy patients aged 2.6 to 20.8 years compared with healthy controls[22] (level III). This may reflect decreased bone strength in cerebral palsy due to smaller and thinner bones. However, assessed by high-resolution magnetic resonance imaging, distal femur trabecular bone micro-architecture was found to be markedly underdeveloped in children with cerebral palsy compared to healthy controls[21] (level IIa).

Conclusion no. 1a

There are sufficient data in the current pediatric literature to support that there may be significantly decreased bone mass in children with cerebral palsy.

Question no. 1b: What are the Risk Factors for Impaired Bone Mineral Density in Children With Cerebral Palsy?

The factors that were found to best correlate with the degree of bone mineral density impairment in children with cerebral palsy were feeding difficulties with low nutritional status and low weight *z* scores, the child's motor functional ability, and prolonged immobilization[12-16,23-27] (level III). Adequate nutrition is an important factor known to influence bone accrual in children.[5,7] Mechanical loading has a critical role in the formation and maintenance of healthy bones; it is essential for normal skeleton development, for longitudinal growth, and for modeling and accumulation of bone mass. Physical activity, particularly intense activity with biomechanical loading, is of the most significant modifiable

factors affecting bone accrual in healthy children.[7,9,28,29] Mechanical loading is influenced by both external forces and muscle action. In cerebral palsy, lack of weight bearing as part of everyday routine and impaired muscle function does not allow bones to be exposed to the mechanical forces needed for their optimal development and growth. Periods of immobilization due to orthopedic and other surgery, following fractures and for various other causes, further enhance this problem.

Another risk factor was antiepileptic drug usage,[12] being used by about 35% of children with cerebral palsy.[1] Drugs that induce the cytochrome P450 enzyme, such as phenytoin, phenobarbital, and less decisively carbamazepine, were shown to impair bone mineralization in children[30,31] (level III),[32] (level IV). There is also evidence of reduced bone mineral density in children treated with valproate, oxcarbazepine, or lamotrigine[30,31,33,34] (level III). Correlations of less significance were found between bone mineral density and calcium intake or short periods of immobilization.[12,13,35]

Growth hormone, insulin-like growth factor,[1] thyroid stimulating hormone, and sex steroids are all important for bone growth in healthy children[7]; however, we found little data dealing directly with the relations between the hormonal status of children with cerebral palsy and their bone density.

Disordered puberty and growth retardation were described in children with cerebral palsy[1,36,37] (level III) and might cause bone mineral density to be lower than expected for age; if unaccounted for, this might lead to misinterpretation of bone mineral measurements.[4,38,39]

Conclusion no. 1b

Children with cerebral palsy that demonstrate low body mass index, have feeding difficulties, are classified as low motor functional ability, demonstrate delayed puberty and growth, and use antiepileptic drugs are at increased risk for low bone mass.

Question no. 2a: Are Children With Cerebral Palsy at Increased Risk for Bone Fractures?

Fracture risk in the general pediatric population is difficult to assess, and variable rates have been reported between 1.33 and 3.6 per 100 person years[40,41]; the most common site for fracture is the radius/ulna and most fractures are attributed to trauma during physical activities. Studies in children with moderate to severe cerebral palsy found the prevalence of fracture history was about 15.5%[12,42] (level III) and following proximal femoral osteotomy surgery, fracture rate was found to be 20%[43] (level III). Fracture incidence in children with cerebral palsy was found to be 4.8 fractures per 100 person years in one study[42]; and 2.98 fractures per 100 person years in another[44] (level III). The most common sites for fractures in children with cerebral palsy were the lower limbs, in particular the femur and tibia[12,44-46] (level III); however, when studying characteristics of fractures in a group of institutionalized, nonambulatory children and young adults 6 to 29 years old, with cerebral palsy and rickets, a dominance of upper extremity fractures was found[47] (level III). The cause for fractures in children with cerebral palsy was often unknown or attributed to minimal trauma and diagnosis was frequently delayed[44,45] (level III).

Several risk factors for fractures were reported: a history of a previous fracture, a feeding gastrostomy tube, higher body fat, older age, rickets, antiepileptic drug treatment, seizures not related to the antiepileptic drug treatment, hip spica casting, a mixed muscle tone disorder, and usage of standing equipment in physical therapy[13,42,44-47] (level III). The severity of ambulatory status impairment was shown to be a risk factor for fractures in a study of a heterogenic group of cerebral palsy children[45] (level III) but not when only moderate to severe cerebral palsy patients were investigated[42] (level III).

Conclusion no. 2a

Based on these data, a simple comparison of fracture rates between the cerebral palsy population and healthy children might be inappropriate. However, given the unique characteristics of their fractures, lifestyle, and risk factors, we suggest children with cerebral palsy who demonstrate one or more of those risk factors should be monitored frequently for symptoms of restlessness, limb deformity, and signs of pathological fractures.

Question no. 2b: What is the Relation Between Bone Mineral Density and Fractures in Cerebral Palsy?

The causal possible relationships between fracture risk and decreased bone mass in the general pediatric population are still debatable; however, a recent meta-analysis suggested such a relation exists.[48,49] King et al[17] reviewed children and adults with spastic cerebral palsy; patients with a history of fractures were characterized by significantly lower lumbar spine bone mineral density dual-energy x-ray absorptiometry *z* scores (level III). Henderson et al reported different results in 2 studies. Initially they did not find lumbar and femoral bone mineral density dual-energy x-ray absorptiometry *z* scores significantly related to a history of previous fractures in children with spastic cerebral palsy[13] (level III); later, as part of the North American Growth in Cerebral Palsy project, they reported fracture history to be related to distal femur bone mineral density *z* scores but not to spine *z* scores[12] (level III). Two studies used quantitative ultrasound to evaluate propensity for bone fractures in children and young adults with severe cerebral palsy[23,50] (level III); quantitative

ultrasound values related to the current existence of fractures and to a history of previous fractures.

A possible relation between bone mineral density and fractures as well as a diagnosis of osteoporosis might be masked by the occurrence of undiagnosed fractures in this population; Shaw[51] suggested considering performing spine X rays to identify asymptomatic vertebral compression fractures when reduced bone density is found in a child with cerebral palsy (level IV).

Conclusion no. 2b

We believe that even though there is no conclusive evidence regarding the relation between bone mineral density and fractures in these children, a causal association is possible. Thus, in a child with cerebral palsy and the suggested risk factors a high index of suspicion concerning fractures should be kept. Additionally, a child with cerebral palsy suffering bone pain or a fracture after minor trauma should be strongly considered for bone mineral density evaluation.

Question no. 3a: What Methods are Available for Assessing Bone Health in Children With Cerebral Palsy?

Bone density. There are several noninvasive methods for assessing bone density in children; these are based on x-ray, ultrasound, or magnetic resonance and none measures bone strength directly. Therefore, all need to relay on estimations of bone density and microarchitecture combined with geometry.

Dual-energy x-ray absorptiometry. Dual-energy x-ray absorptiometry is currently the method of choice for assessing bone mineral density. It analyzes the attenuation of x-rays of 2 energies transversing through bone to estimate its density and mass.[5] Results can be presented in several ways: bone mineral content in grams, bone mineral density, or areal bone mineral density in grams per cm^2, alculated by dividing the bone mineral content by bone area, volumetric bone mineral density in grams per cm^3 considering thickness as well, and bone mineral density *z* scores.[52] Bone mineral density *z* score is the standard deviation from age- and gender-specific norms. A score below −2 is considered low bone mass.[3,4,8]

Dual-energy x-ray absorptiometry is a rapid, easy to use, accurate, and reproducible method; however, it has a number of limitations in children: (*a*) being a 2-dimensional technique, dual-energy x-ray absorptiometry does not measure a true volumetric density and results are influenced by bone size and shape.[5,48,53] In growing children, bone dimensions of different regions of the skeleton change consistently, in relation to the child's age, growth rate, and pubertal stage[7] (level IV), while the bone mineral density *z* scores consider average height and weight during youth. This might cause inaccuracy of bone mineral density assessment in children with growth impairment as might be the case in children with cerebral palsy.[38] The reported delay in skeletal maturation and growth as well as possible precocious puberty reported in patients with cerebral palsy may cause significant inaccuracy in bone mineral density assessment[1,36,37] (level III) and adjustment of results with height, skeletal age, and pubertal stage has been suggested.[4,8,38,39,48] (*b*) Dual-energy x-ray absorptiometry assumes homogeneity in the composition of soft tissues near the bone; however, this composition and specifically the proportions of fat versus lean tissue are unknown and variations were shown to cause major inaccuracies.[6,52] (*c*) In lumber spine assessments, posterior vertebral elements included in the scan might increase bone mineral density estimations.[52] (*d*) Reaching the machine is a limiting factor for severely disabled children and positioning might be compromised due to movement, flexion contractures, and other skeletal deformities.[12,13,23,25,27] (*e*) Changes associated with surgery, osteotomy, or metallic fixation devices might impair interpretation.[8] (*f*) The radiation exposure makes dual-energy x-ray absorptiometry less appealing as a tool for screening or routine follow-up. (*g*) Bone density at different sites is not equally affected by the same extrinsic and intrinsic factors, which may be partially attributed to the different proportions of trabecular and cortical bone.[5,48] When assessing dual-energy x-ray absorptiometry *z* scores of spine and femur in children with cerebral palsy, significant differences between *z* scores of the 2 sites have been found as well as different relations to various parameters including fracture history[12,44] (level III),[54] (level IIa). The usage of whole-body dual-energy x-ray absorptiometry bone mineral content has benefits, which may overcome some of these difficulties.[39] It includes all skeletal regions, including trabecular and cortical regions, has adjustments for height variability, is easy to complete, and involves low radiation, though a longer scanning time.[5,53] Because the skull has a major influence on results, it is often not included in whole-body dual-energy x-ray absorptiometry, especially in young children. Lumbar spine, whole-body bone, and proximal femur are the commonest sites tested by dual-energy x-ray absorptiometry in children for which reference values are available.[4,5,8,54] A method of measuring bone mineral density at the distal femur by dual-energy x-ray absorptiometry in children with cerebral palsy has been described and used successfully[12,54-56] (level IIa, III). The International Society for Clinical Densitometry considers spine and whole-body less head bone mineral content and areal bone mineral density to be the most accurate and reproducible in children and recommends performing these scans in children with chronic immobilization at fracture presentation.[8]

Quantitative computed tomography. This is a 3-dimensional technique, giving true volumetric measurements (volumetric bone mineral density); quantitative computed tomography can assess bone size and shape and differentiate between cortical and trabecular bone.[4,48,52] In this technique, the attenuation of x-rays transmitted through the bone in various directions is analyzed. Spine and tibia quantitative computed tomography have been used in children with cerebral palsy[57,58] (level Ib),[15,25] (level III).

Limitations include (*a*) lack of complete normative pediatric database[4,48]; (*b*) the high radiation involved; (*c*) a significant cost-accessibility difficulty; and (*d*) possible interference caused by movement and metallic devices.[5,53] Peripheral quantitative computed tomography[4,48] holds the advantages of quantitative computed tomography with less radiation involved and a shorter scanning time. It is common practice to use a single scan; however, this may reduce reproducibility. In a study analyzing peripheral quantitative computed tomography data from children with cerebral palsy, a large variability in the profiles of decay in metaphyseal trabecular bone density along the length of the proximal tibia was demonstrated, indicating that a minimal offset in positioning of a slice would cause significant errors[59] (level III).

Quantitative ultrasound. In this technique, ultrasound transducers quantitate the transmission velocity through the bone evaluated as well as the signal attenuation.[5] Quantitative ultrasound is presumed to assess parameters determined by the amount of bone and the bone's material and structural properties.[4,48,60] Normal values according to age and gender exist. In addition to not involving ionizing irradiation, its simplicity of use, short test time, lower cost, and the considerably small and portable device make it a useful tool for usage in the clinic and for testing bedridden children who are difficult to mobilize for dual-energy x-ray absorptiometry.[5,23,48] Quantitative ultrasound of the calcaneus, tibia, radius, and phalanxes have been assessed in children with cerebral palsy[38] (level IIa),[23,25,50] (level III). Limitations of this method are (*a*) technical problems due to very thick soft tissue or severe agitation[23]; (*b*) the limited experience in children; (*c*) measurements might be affected by temperature[52,61]; (*d*) although quantitative ultrasound has been evaluated in comparison to bone mineral density measurements in both healthy children and children with various chronic illnesses, there is lack of reproducibility.[36,60,62-66]

Magnetic resonance imaging. Magnetic resonance imaging uses the principles of magnetism to create 3-dimensional images. This method is based on measuring a signal generated by the magnetic field of the hydrogen nuclei; it produces a negative image of the bone matrix and a positive image of the interstitial space between trabeculae.[67,68] High-resolution magnetic resonance imaging, also named micromagnetic resonance imaging or "virtual bone biopsy," is a complex process detecting bone microarchitecture. Magnetic resonance imaging holds the advantage of not exposing subjects to irradiation and providing various cross sections without moving the patient. Its reproducibility in children with cerebral palsy was very good[21] (level IIa). Its major limitations are (*a*) an age limitation: magnetic resonance imaging is optimal when cancellous bone is surrounded by fatty marrow and thus less optimal in children; (*b*) it cannot be used for children with metallic devices, as many cerebral palsy children are operated; (*c*) long scan durations; and (*d*) subject motion can interrupt imaging.

Biochemical turnover markers. Specific markers of bone formation are serum bone specific alkaline phosphatase, osteocalcin, carboxyl (C)-terminal extension of procollagen type I, and the amino (N)-terminal extension of procollagen type III. Markers of bone resorption are urinary hydroxyproline, hydroxylysine, *N*-telopeptide, pyridinoline, deoxypyridinoline, carboxyterminal telopeptide of type 1 collagen and bone specific hydroxypyridinium collagen cross links.[5,69]

Various bone markers were measured in children with cerebral palsy, yet results are both sparse and inconsistent; osteocalcin levels were suggested to have inverse correlations with lumbar spine bone mineral density *z* scores and age in children with spastic cerebral palsy,[17] however, a nonreproducible finding so far.[12] Pluskiewicz et al[27] found bone alkaline phosphatase to be lower in immobilized patients, most of them with cerebral palsy, compared to controls[27]; carboxyterminal telopeptide of type 1 collagen correlated with forearm bone mineral density in these immobilized patients.

Question no. 3b: Which is the Most Applicable Assessment Method in Children With Cerebral Palsy?

Dual-energy x-ray absorptiometry is the most widely used and preferred technique for assessing bone mineral density in children with cerebral palsy today and most data presented are based on this method.[8,12-14,16-18,20,26,44,70-75] Quantitative computed tomography and peripheral quantitative computed tomography have also been used in children with cerebral palsy[15,22,50,58] and are considered accurate however considerably less accessible. We found 1 study using high-resolution magnetic resonance imaging in children with cerebral palsy; reproducibility was very good[21] (level IIa). Few studies evaluated the reliability of quantitative ultrasound in children with cerebral palsy[23,25,27,50] (level III).

Conclusion no. 3b

Currently, dual-energy x-ray absorptiometry offers the best combination of cost, ability to adjust for body size, and availability despite its limitations. To increase its accuracy regarding adjustment for growth and puberty and overcome difficulties, we suggest whole body is preferable. We think more reference data should be gathered regarding quantitative ultrasound, peripheral quantitative computed tomography, and distal femur dual-energy x-ray absorptiometry prior to using them regularly. Magnetic resonance imaging and quantitative computed tomography do not seem feasible to us in daily life context. Measurement of blood and urine bone and specific markers is used to evaluate bone turnover; this can aid in determining the pathophysiology of reduced bone strength. Large-scale studies should still be performed to elucidate the role of bone turnover markers in the pediatric population in general and in this subset of children, and they are not yet applicable as a monitoring or survey method.

Question no. 4: How Can Bone Strength Be Improved in Children With Cerebral Palsy?

Physical activity and load bearing. Possible benefit from physical activity and load bearing among children with cerebral palsy was suggested by small randomized controlled studies[57,58,71] (level Ib). Prolonging standing duration in children with cerebral palsy improved vertebral volumetric bone mineral density by 6%; however, tibial volumetric bone mineral density did not change.[57] Load-bearing physical activity increased significantly femoral bone mineral content by 9.6% to 11.5% and estimated volumetric bone mineral density 5.6%. A six-month program of short daily doses of low magnitude, high-frequency loading (vibration) among ambulant children with disabling conditions demonstrated a significant increase in tibial volumetric bone mineral density, with a net benefit of 17.7%; however, compliance was low.[58]

Vitamin D and calcium supplementation. Two studies investigated this treatment in children with cerebral palsy. One study found vitamin D supplementation effective in decreasing fracture rate in children with severe cerebral palsy and rickets[47] (level III). The other study demonstrated supplementation of vitamin D and calcium to children with severe cerebral palsy and epilepsy treated with various antiepileptic drugs to be associated with a significant increment in bone mineral density[74] (level IIa); however, vitamin D levels were not measured. We would like to emphasize that routine administration of calcium and vitamin D to any child with low bone density has not proved to be beneficial[51]; if there is evidence of vitamin D deficiency or poor dietary calcium intake, replacement would be appropriate.

Bisphosphonates. Bisphosphonates have been suggested to improve bone status[19,70,72] and reduce fracture risk[8,9] in children with cerebral palsy. Intravenous pamidronate significantly increased bone mineral density in children with severe cerebral palsy and reduced bone density[72] (level IIb), in a small group of children with quadriplegic cerebral palsy (femoral z score -4 vs -1.8)[70] (level Ib), and in children with cerebral palsy with a history of fractures and abnormally low bone mineral density (spinal z score -4.1 vs -2.5)[19] (level III). Fracture rate decreased from 0.34 to 0.04 fractures per person years[19] (level III). During post-treatment follow-up of 2 to 3 years of those patients, average metaphyseal distal femur bone mineral density z score decreased from a mean of -1.7 to -3.6 but with a reduced fracture rate[18] (level III). In a randomized double-blind study of 20 children with cerebral palsy and osteoporosis, those treated with vitamin D and a third generation bisphosphonate (risedronate) had a larger increase in bone mineral density compared with children treated with vitamin D alone[76] (level Ib).

Growth hormone. Insulin-like growth factor 1 and insulin-like growth factor–binding protein 3 were found to be decreased in children with cerebral palsy[20] (level III); in those having a bone mineral density z score under -1 SD, a significant correlation between insulin-like growth factor-binding protein 3 levels and bone mineral density was demonstrated and the correlation between insulin-like growth factor 1 and bone mineral density was near significance. These findings support the hypothesis that the growth hormone-insulin-like growth factor axis is impaired in these children. Five children with cerebral palsy and decreased spinal bone mineral density were treated with recombinant growth hormone for 18 months[73] (level Ib). Average bone mineral density z score increased significantly by 1.17 SDs in the treatment group compared to no significant change in the control group.

Vitamin K. Vitamin K has been proposed as a promoting bone formation factor.[37] Fifteen months of treatment with a combination of vitamin D and vitamin K2 to a small series of institutionalized bedridden prepubertal children, treated with antiepileptic drugs, showed significant increase in serum osteocalcin, in relative rate of bone formation/resorption markers and in the z score of cortical bone mineral density of second metacarpal bone[77] (level III). In a case report of a child with hemiplegia treated with vitamin K alone, the cortical bone geometric strength of the hemiplegic tibia increased compared with the non-hemiplegic tibia[78] (level IV).

Conclusion no. 4

Physical activity, especially programs implying biomechanical loading, is the only suggested modality to be safely

recommended when low bone mass is detected without any history of pathological fractures or spinal compression fractures (asymptomatic low bone mass). Bisphosphonate treatment should be considered in children with cerebral palsy and a history of fractures and abnormally low bone mineral density.

Conclusion

In this review, we addressed the short-term aspects of bone health in cerebral palsy. Most data are derived from nonrandomized descriptive studies, not based on large populations; some retrospective data at an evidence level III are available, although not plentiful, and we found only a few level II studies.

We conclude that the topic of bone health in children with cerebral palsy should be regularly emphasized and meticiously followed and addressed during the multidisciplinary team follow-up in the clinic; specific attention should be paid to risk factors such as previous fractures, periods of immobilization, bone pain, lack of mechanical load, and low motor ability. Currently, the best method to detect low bone mass is dual-energy x-ray absorptiometry; however, this technique is not flawless. Even though a number of scanning sites have been assessed in cerebral palsy, we believe whole-body dual-energy x-ray absorptiometry is preferable as it overcomes some of the method's major limitations. Data are lacking regarding the reliability and efficacy of distal femur dual-energy x-ray absorptiometry, peripheral quantitative computed tomography, quantitative ultrasound, and bone markers, and until larger studies are performed they could not be used routinely.

Load-bearing physical activity cannot be overemphasized as primary and secondary prevention. Alertness for detection of pathological fractures and spine compression fractures should be kept and if a fracture is detected in face of low bone mineral density, and the appropriate roentgenographic characteristics, a diagnosis of osteoporosis should be raised and therapy with bisphosphonates may be considered.

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