Review Article

A Comprehensive Review of Bone Health in a Child: From Birth to Adulthood

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ABSTRACT

Bone health is critical for growth and development during childhood. Although fractures are common in children, fractures occurring in the absence of trauma should prompt physicians to consider underlying bone health disorders. This article provides an overview of the current definition of osteoporosis in children, highlighting its limitations and the potential for underdiagnosis. It also discusses the timing of screening initiation and various techniques used to assess bone health, along with their respective benefits and limitations. In addition, this article identifies several causes of primary and secondary osteoporosis in children, shedding light on previously overlooked disorders that can contribute to poor bone quality. The article emphasizes the importance of a multidisciplinary approach to therapeutic management and aims to optimize patient outcomes and improve the overall care of pediatric bone health disorders.

The orthopaedic team is one of the first groups notified when a child sustains a fracture. In this setting, the orthopaedic surgeon must recognize key features of the case to determine the fracture's etiology, and whether it suggests underlying bone fragility that might warrant additional work-up.

Identifying children early on who are not optimizing their bone health is imperative because they face heightened susceptibility for subsequent fractures and a poorer quality of life. Diagnosing pediatric osteoporosis is complex and relies not only on acknowledging the definition of pediatric osteoporosis but on discerning clinical findings, using laboratory studies, and in certain cases using molecular genetic testing to ascertain whether a disorder exists that could impair bone quality. Recognizing the at-risk child early on is a major advantage because the pediatric skeleton has the potential to improve with the appropriate treatment. With increased awareness of childhood osteoporosis, the orthopaedic surgeon can quickly intervene and coordinate a multidisciplinary team response which can have a dramatic and positive effect on long-term bone health.

Normal Bone Health

Bone Health

Bone health is determined by a delicate interplay of factors influencing bone quality including bone density, geometry, turnover, cortical thickness and

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porosity, trabecular bone architecture, and the attainment of peak bone mass (PBM).[1] Bone is comprised of an organic component consisting of cells, collagen, and noncollagenous proteins and an inorganic component composed of hydroxyapatite crystals and minerals. The degree of mineralization is considered an important factor in quality.[1] Bone development begins prenatally, typically around 8 to 12 weeks of gestation, with 80% of fetal bone mineralization occurring during the third trimester.[2] The third trimester is a critical period when enhanced calcium absorption takes place through active placental transport from the mother.[3] Preterm children (<37 weeks) have markedly lower bone mineral density (BMD) than their term counterparts when BMD is measured 7 or 8 years later.[4] Therefore, physicians must stress the importance of good bone health during childhood, especially for children born prematurely.

The skeleton continues to develop throughout childhood; the growth of tubular bones involves lengthening through endochondral apposition and widening through periosteal apposition.[5] Periosteal apposition contributes to the outer diameter of bone, a major component of bone strength.[1] During this phase, rates of bone mineral content and BMD exhibit relatively consistent growth patterns between both sexes.[6]

Throughout the pubertal years, a notable surge takes place in bone mass and BMD. Although the timing of this process differs between male and female patients; usually the rate of peak bone accrual is highest during Tanner stages 3 and 4 also correlating within 6 months to 2 years after peak height velocity.[7] Typically, in female patients, this increase occurs between 11 and 14 years, with a subsequent tapering off 2 to 4 years postmenarche.[5] Male patients experience PBM accrual between 13 and 17 years, although some individuals may continue this process until age 20 years.[5] Recent studies suggest that the attainment of peak bone content may occur even later, around 21 years in female patients and 25 years in male patients.[6]

At the culmination of PBM accrual, female patients often exhibit lower BMD compared with male counterparts. This disparity is attributed primarily to shorter maturation times, smaller bone sizes, and less periosteal apposition in female patients. Overall, reaching PBM in adolescence holds considerable importance as evidenced by epidemiologic research that has shown that a 10% increase in PBM can delay osteoporosis by 13 years and may be the strongest mitigator of future fractures.[8]

Fracture Risk in Children: Identification of Modifiable Factors

Fracture Incidence in Children

Fractures are a common occurrence during childhood, with rates ranging from 100 to 200/10,000 person-years in preschool years and 200 to 450/10,000 person-years during adolescence.[9] Yet, pediatric fractures may represent skeletal fragility, a disorder in which abnormal bone mass, geometry, microarchitecture, and/or growth lead to increased fracture risk.[10] Indeed, studies show that BMD is consistently lower in healthy children with fractures compared with nonfracture control subjects.[11] Moreover, in children with higher risk of fracture, a reduction in one SD in BMD imparts an 80% increased odds of vertebral fracture.[12]

Although low BMD correlates with increased risk of fracture, there are many factors that can help children gain bone mass and reduce fracture incidence during growth. With early recognition and appropriate intervention, children are not destined to track within a low-BMD quantile for the remainder of their growth.[13]

Body Mass Index

Body mass index (BMI) affects PBM. In a longitudinal study conducted in Spain between 2006 and 2013, Lane et al[14] followed a cohort of children for 11 years and found that BMI played a notable role in fracture risk. They found that children with obesity had a 1.26 hazard ratio for any fracture and a 1.74 ratio for lower extremity fractures even after adjusting for age, sex, socioeconomic status, and nationality.

Nutrition

Inadequate dietary intake also plays a role in fracture risk. Goulding et al[15] demonstrated that the risk of fracture was 5 times higher in children who had low dietary calcium consumption. Adolescents with anorexia nervosa have a 60% increased relative risk of fracture compared with age-matched control subjects.[16] Moreover, fractures occurred even without markedly lower BMD scores. This indicates that BMD measurements may not reflect the true risk a child with anorexia has for sustaining a fracture. Together, these studies highlight the stark effects nutrition can have, even in a short time frame, on bone health.

Exercise

Participating in impact-loading exercise is also crucial for bone health in the child and throughout the adult life. Research has shown that the responsiveness of bones to

loading forces during adolescence may be twice as great as the response seen when training begins at a later age.[17] Adolescents participating in high-impact exercise (dance, football, or basketball) have markedly higher BMD in comparison with their nonexercising and low-impact exercising counterparts. The consensus is that impact-loading exercise program performed routinely throughout adolescence are best at stimulating bone strength.[5]

However, children can also overexercise and as a result experience stress fractures or enter an energy-deficit state that can negatively affect bone growth. In a large cohort of girl runners, aged 6 to 18 (average of 14.4 years), a five-fold increase in stress fractures occurred over a 5-year period.[18] The authors suggested early specialization in a single sport can be a major risk factor for fragility fractures in children and a thorough evaluation of exercise duration and type (particularly track & field, dancers, or military recruits) should be done when indicated.

Societal Influences

Societal factors can also contribute to fracture risk. During the COVID-19 pandemic, clinicians observed a decrease in fractures.[19] This decrease was attributed to reduced participation in sports activities because of lockdowns and social restrictions. However, no changes were seen in fracture incidence in children aged younger than 3 years, who often experience fractures due to falls.

Other Factors

Breastfeeding at a young age and having lean body mass have been shown to push children into higher BMD quantiles.[4,13] Furthermore, smoking and hormone levels contribute to fracture incidence.

These examples demonstrate a multitude of modifiable factors contributing to childhood fracture rates. Therefore, an astute physician has the opportunity to intervene in any or all of these factors, offering assistance to children that may be tracking in lower BMD quantiles.

Who Should Have a Comprehensive Bone Health Evaluation?

Determining who should undergo a comprehensive bone health evaluation relies on understanding the fracture severity, supporting clinical evidence, and risk factors for bone fragility. Identifying the sentinel fracture or one that signifies risk for recurrent fractures can be extremely valuable for the child's long-term health.

Figure 1 shows a proposed approach to the child with a fracture. As no definitive method exists, this suggested systematic approach is based on the recommendations of Ward et al and Ciancia et al[20,21] These authors advise assessing the fracture morphology. This can be done by "grading" the fracture based on its location (eg, vertebra, femur, humerus, or flat bones may be indicators of poor bone strength) and mechanism (high or low trauma defined as fall from standing height or at walking speed). Physical findings such as blue sclera, joint hypermobility, teeth alterations, known risk factors such as chronic glucocorticoid use, or a positive family history indicate health conditions that may predispose the child to fracture.[20,21] This approach should trigger a more thorough bone health evaluation.

In cases in which clinical suspicion arises, healthcare providers should then (1) rule out a disorder of mineral metabolism and (2) evaluate for an acute or chronic systemic illness. A thorough history should include diet, weight loss, fatigue, stool pattern, menstruation, and exercise habits. Primary laboratory tests include a complete blood count with differential, comprehensive metabolic panel, erythrocyte sedimentation rate/C-reactive protein, 25-hydroxy vitamin D, T4/thyroid-stimulating hormone, parathyroid hormone, magnesium, phosphorous, and spot urinary creatinine and phosphate. A physical examination looking for long bone bowing, craniotabes, or widening of the ankles or wrists can help clinicians diagnose disorders of mineral metabolism. Additional laboratory studies or genetic testing may be considered as well. Symptoms such as weight loss or fatigue may trigger an evaluation for systemic illnesses. Finally, additional radiographs can help if there is suspicion of structural abnormalities.

If these initial assessments yield negative results, clinicians should conduct additional evaluation for osteoporosis. This is done preferably through dual x-ray absorptiometry (DXA), which remains the benchmark, and less frequently with a lateral thoracolumbar spine radiograph.[20]

Osteoporosis in Children

Definition

The definition of osteoporosis in the child has evolved over the last decade. In 2013, the International Society for Clinical Densitometry (ISCD) focused on creating a definition of osteoporosis to prevent overdiagnosis. In 2019, the ISCD further revised the definition; it now

Figure 1

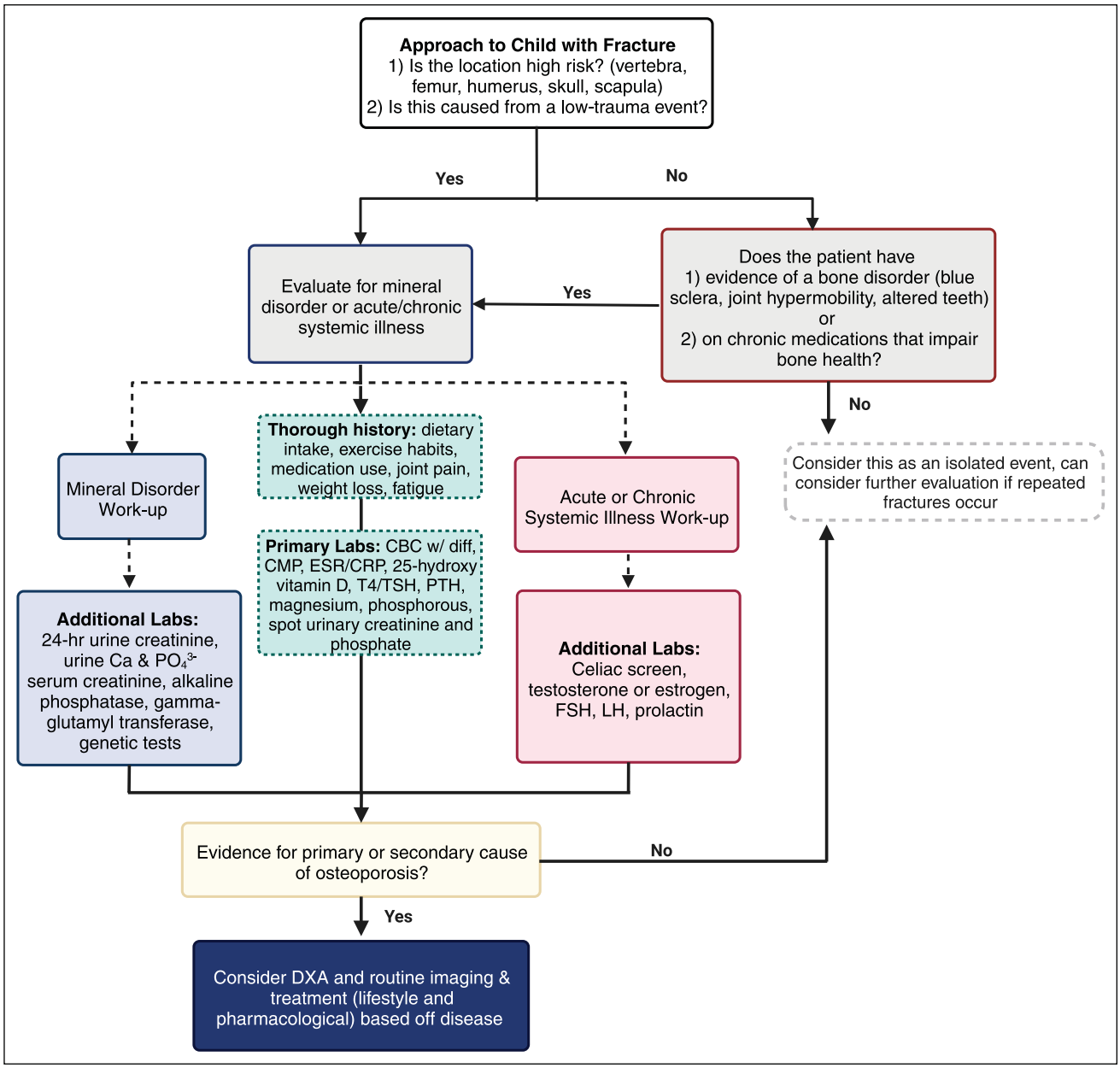

Diagram showing an approach to the child with a fracture. Ca = calcium, CBC = complete blood count, CMP = comprehensive metabolic panel, ESR/CRP = erythrocyte sedimentation rate/C-reactive protein, FSH = follicle-stimulating hormone, LH = luteinizing hormone, PO_{43-} = phosphate, PTH = parathyroid hormone, T4 = thyroxine, TSH = thyroid-stimulating hormone

requires the presence of both a clinically significant fracture history and BMD Z-scores ≤ −2.

The ISCD defines that a clinically significant fracture history is one or more of the following: (1) two or more long bone fractures by age 10 years and (2) three or more long bone fractures at any age up to 19 years. In addition, the finding of one or more vertebral compression fractures is indicative of osteoporosis, in the absence of local disease or high-energy trauma, regardless of the BMD Z-score.[22]

Definition Limitations

However, this definition has limitations because it does not consider all characteristics of skeletal fragility, such as key radiographic findings like radiolucent lines in weight-bearing regions or certain exposures like

osteotoxic drugs, that may indicate poor bone health.[20] As such, critiques of the ISCD definition state that it may lead to underdiagnoses. These are patients for whom early recognition and treatment might have led to recovery and the opportunity to reshape a deformity to a more anatomic alignment.

Importance of Identifying Osteoporosis

Children with certain health conditions, such as chronic glucocorticoid use, osteogenesis imperfecta, neuromuscular disorders, or leukemia, may not meet the defined threshold for an osteoporosis diagnosis but can still sustain fractures even with Z-scores above −2.0.[23] Therefore, waiting for another long bone fracture or low BMD to meet the ISCD criteria may delay necessary osteoporosis treatment. With early identification and recognition of underlying pathology, healthcare teams can help identify high-risk children to avoid the adverse health outcomes associated with fractures, such as transient disability, pain, and loss of height.[21]

Causes of Osteoporosis

Low BMD and increased fracture risk in children are associated with various conditions, which can be classified as primary or secondary causes of osteoporosis. Primary osteoporosis refers to conditions of genetic origin, as noted in Table 1; secondary osteoporosis arises from chronic conditions or their treatments, appearing in Table 2.

Primary Osteoporosis

The most prevalent primary cause of childhood osteoporosis is osteogenesis imperfecta (OI), characterized by abnormalities in type I collagen. OI has an incidence of approximately one in 15,000 to 20,000.[24] Clinical features can also include dentinogenesis imperfecta, blue sclerae, ligamentous laxity, and hearing loss. The phenotypic range of OI is broad, making genetic testing highly valuable for diagnosis.

Table 1. **Major Causes of Primary Osteoporosis**

Osteogenesis imperfecta (OI)
Marfan syndrome
Ehlers-Danlos
Metabolic disorders
Homocystinuria
Wilson disease
Menkes syndrome
Galactosemia
Hypophosphatemic rickets
Idiopathic juvenile osteoporosis

Hypophosphatemic rickets is another cause of primary osteoporosis. It is characterized by low levels of serum phosphate, leading to impaired bone mineralization and skeletal abnormalities.[25] This condition can be hereditary or acquired and is often associated with mutations in genes involved in phosphate regulation. Early diagnosis and appropriate management of hypophosphatemic rickets are crucial in preventing long-term skeletal complications and optimizing bone health.

Idiopathic juvenile osteoporosis is a diagnosis of exclusion and is typically observed in prepubertal children who present with hip, back, and lower limb pain, along with fractures.[26] In these patients, there are typically no extraskeletal findings or growth impairment, and often there is full resolution of symptoms by puberty. However, ruling out identifiable genetic forms of osteoporosis by advanced molecular genetic tests is recommended.

Secondary Osteoporosis

The common causes of secondary osteoporosis include chronic inflammatory disorders, chronic immobility, iatrogenic causes (mainly glucocorticoid use), leukemia along with other childhood cancers, hematologic disorders, and nutrition disorders.[21] Inflammation and cancer can upregulate proinflammatory cytokines, which may induce bone resorption and inhibit formation.[27] These conditions can also impair the growth hormone insulin-like growth factor I axis, which is essential for achieving PBM.[25] The severity of secondary osteoporosis is highlighted by studies showing that vertebral fractures have been found in 33% of children with leukemia and 25% of children with neuromuscular disorders.[28]

Osteotoxic medications are also a leading cause of secondary osteoporosis. One of the readily used ways to treat disorders of secondary osteoporosis, such as inflammatory disease and Duchenne muscular dystrophy, is glucocorticoids, which are osteotoxic drugs. Studies have found that in myopathic diseases, the prevalence of vertebral fractures reaches more than 50% while on corticosteroids.[29] In another study, there was a five-fold increased risk of vertebral fractures for every 10 mg/m^2 increase in average daily glucocorticoid dose in children with leukemia.[30] Therefore, it is crucial for physicians to understand how both the underlying disease and its treatment can contribute to fracture risk.

Table 2. Major Causes of Secondary Osteoporosis

Inflammatory disorders
Inflammatory bowel disease
Cystic fibrosis
Rheumatic disorders
Chronic immobilization
Cerebral palsy
Myopathic diseases
Endocrine disturbances
Turner syndrome
Type 1 diabetes mellitus
Thyroid disorders
Hypogonadotropic hypogonadism
Hyperprolactinemia
Hyperparathyroidism
Cushing syndrome
Growth hormone deficiency
Cancer and corresponding therapies with adverse effects on bone
Hematologic disorders
Sickle cell disease
Thalassemias
Osteotoxic agents
Glucocorticoids
Antiepileptics
Anticoagulants
Methotrexate
Antiretrovirals
Medroxyprogesterone
Hormonal suppression
Nutrition/malabsorption
Anorexia
Avoidant restrictive food intake disorder
Calcium/vitamin D malabsorption or deficiency
Celiac disease

This enables physicians and families to weigh the risks and benefits of different treatment modalities.

In recent years, providers are more frequently recognizing poor bone health related to disorders of nutrition and energy; these include obesity, overtraining in sports, avoidant restrictive food intake disorder, and anorexia nervosa.[31] Poor dietary intake is common in children with anxiety, learning disorders, autism, and other mood disorders; these are cases where providers should have a lower threshold for obtaining a thorough nutrition history.[32]

The effect of medical therapies for transgender care on bone health is relatively unknown. Early data suggest that children and adolescents undergoing pubertal suppression for gender dysphoria are at risk for delayed BMD accrual. In 1 year, adolescents on gonadotropin hormone-releasing hormone therapy can drop into osteopenic Z-score ranges, although height continues to increase.[33]

Identifying the interplay between these disorders, nutrition, hormonal balance, and energy balance will help identify many children at risk of fractures who were previously overlooked.

Techniques for Assessing Bone Health

Imaging Techniques

Various imaging techniques are available to assess bone health (Table 3). The most preferred technique is a DXA scan. DXA machines are widely available, quick, and emit low radiation. DXA scans provide two-dimensional projections that yield areal BMD measurements. In adults, DXA scans focus on the distal radius, femur, and hip, while pediatric DXAs are done on different anatomic locations notably the AP lumbar spine and total body less head. In unique cases, such as cerebral palsy, the distal femur is used for both technical feasibility and its clinical relevance to fractures.[23] These measurements can be extrapolated to estimate true volumetric BMD in g/cm^3.

In children, interpretation of DXAs provides Z-scores, which compare the results with age-matched control subjects rather than with healthy adults. However, interpreting DXA results can still be challenging because of variations in the reference database used.[20] Recent improvements have been made in pediatric reference databases to enhance accuracy. The Bone Mineral Density in Childhood Study formulated adjusted Z-scores based on height, age, sex, and race to improve BMD interpretation for Hologic systems.[34] Advances in DXA imaging have also allowed for high-quality spine images, enabling the identification of vertebral fractures without the more notable radiation exposure from plain radiographs.[35] In fact, the 2019 ISCD executive summary states that DXA vertebral body assessment may be used as a substitute for radiographs.

Radiographs are obtained when there is clinical suspicion of fracture. Radiographs are valuable for assessing vertebral fractures using modified Genant methods,

Table 3. Imaging Techniques for Assessing Bone Health

Imaging Technique	Description
Dual-energy x-ray absorptiometry (DXA)	Two-dimensional projections providing areal bone mineral density (BMD) measurements, estimation of volumetric BMD in g/cm^3, Z-scores for interpretation, potential underestimation/overestimation of BMD, advances in spine imaging for vertebral fracture assessment
Plain radiographs (x-rays)	Correlation with BMD, evaluation of thin cortices and vertebral fractures, useful for diagnosing and monitoring osteoporosis
Quantitative CT	True volumetric BMD measurements, central or peripheral, limited by reference data and standardized techniques
MRI	Research tool for analyzing trabecular and cortical bone, used when suspicion for fracture is high, avoids ionizing radiation

which define vertebral fractures as >20% height collapse.[21] As vertebral fractures are included in the current definition of osteoporosis, radiographs are an important diagnostic and monitoring tool in high-risk individuals.

Imaging modalities typically used for research include CT and MRI. High-resolution peripheral quantitative CT evaluates the microarchitecture of separate cortical and trabecular compartments. However, the lack of both standardized scan techniques and reference data limits the use of CTs. MRI-based imaging is used primarily in research to analyze trabecular bone volume, cortical bone quantity, and quality.[35] In clinical settings, MRI may be used when suspicion for fracture is high but indeterminate on radiography.

Dynamic Techniques

Dynamic techniques provide unique insights into bone metabolism. However, they are rarely used in clinical practice. They include serum bone turnover markers and transiliac histomorphometry. Bone turnover markers may fluctuate by sex, age, exercise, or meals limiting their ability to provide insight into bone turnover.[35] Transiliac histomorphometry uses tetracycline labeling with harvesting from iliac bone. Previously, transiliac biopsies helped characterize the mechanistic effects of antiresorptive medications and gave evidence of hereditary forms of poor bone health before advanced genetic testing.

Management of Osteoporosis

Deciding Who to Treat

Once the diagnosis of osteoporosis has been made, clinicians and their patients can consider different levels of treatment—ranging from surveillance to pharmacologic support. Treatment is specific to the disease etiology: Whether the condition is transient, age at diagnosis, pubertal status, and current medications all factor into the decision. In many cases, lifestyle modifications are adequate; in other cases, the disorders warrant pharmacologic interventions. Ward[36] described an approach for when to initiate monitoring for secondary osteoporosis and possible next steps, including obtaining lateral spine imaging and monitoring BMI and BMD typically every 2 to 3 years. These results demonstrate that even in the same disease, clinicians can do take different approaches to treatment.

Multipronged Approach

A multipronged approach to managing children with osteoporosis is necessary. A team of physicians (orthopaedic surgeons, pediatricians, endocrinologists, and geneticists), physiotherapists, psychologists, and nutritionists may all be involved in the treatment plan.[25,28]

Optimizing nutritional status is crucial, and high-impact weight-bearing activities and resistance exercise program 2 to 3 times a week should be promoted when feasible. Other physical therapies tailored to specific conditions may be recommended, such as whole-body vibration for children with spastic diplegias or thalassemia. Monitoring and optimizing vitamin D status is important, especially when considering certain medications such as bisphosphonates, because transient hypocalcemia can occur if vitamin D and calcium levels are not preemptively optimized. Children with inflammatory diseases may benefit from steroid-sparing modalities, such as infliximab for rheumatoid disease or inflammatory bowel disease, which have shown positive associations with bone formation markers as well as trabecular BMD and cortical area Z-scores.[27]

Bisphosphonates

In primary causes of osteoporosis, the threshold to treat with medical therapeutics is lower than for secondary osteoporosis; the main reason is that the removal of causative factors is impossible. In these cases, bisphosphonates, which act to reduce bone resorption, are often started. Pamidronate and zoledronic acid, both administered intravenously, are the preferred choice in children because of their rapid infusion and better tolerability compared with oral forms.[25] In OI, a subsequent halving of the annual dosage can be done if the Z-score is improved to within the range of −2.0 to 0 or complete cessation if Z-scores normalize.[37] An annual review of BMD should be done while on these agents.

For secondary osteoporosis, many children do not require bisphosphonates to achieve complete recovery. Ward et al suggested categorizing children into three risk groups based on the duration of their condition: (1) with transient bone health threats (leukemia), (2) with variable bone health threats (inflammatory diseases), and (3) with permanent bone health threats (neuromuscular diseases).[38] Diphosphonate treatment may be considered if there are signs of early vertebral collapse or low–trauma-associated long bone fractures, especially in patients who are postpubertal, will be on more than 3 months of steroids, have impaired mobility, or suffer poorly controlled disease.[38]

Randomized trials have demonstrated that intravenous zoledronic acid can markedly increase lumbar spine BMD in 1 year. In children with glucocorticoid-induced osteoporosis, zoledronic acid increased BMD Z-scores by 0.58 compared with placebo.[39]

Yearly reassessments should be done to determine whether to continue therapy. These assessment should include repeat DXA scans, determining if the child has ongoing fractures or bone pain, their mobility, and other medication use.[37]

Atypical femoral fractures are an extremely rare occurrence with bisphosphate use in children. Thus, the benefit of their use to prevent severe fractures is regarded to outweigh the risks for these rare cases. Notwithstanding, these skeletal agents are used on an off-label basis.

Denosumab

Denosumab is a monoclonal antibody that targets receptor activator of nuclear factor kappa-beta ligand and has been used off-label in limited cases of childhood osteoporosis or sometimes after failed treatment with bisphosphonates. However, physicians must use extreme caution because of the risk of notable rebound hypercalcemic episodes when the drug is discontinued.[28] Ongoing studies are in progress, including a phase III trial to determine effects in glucocorticoid-induced osteoporosis (NCT03164928).

Anabolic Agents

Other studies have investigated using anabolic agents, such as growth hormone, testosterone, and estrogen, to improve bone health in children. In boys with delayed puberty, especially common in Duchenne, using testosterone as an anabolic agent can help achieve higher PBM during this critical period of growth.[28] In female patients with anorexia nervosa, estrogen replacement has been shown to be helpful in improving BMD. Of note, studies suggest that the benefit is only seen with transdermal estrogen as opposed to the oral form, which is important for physicians to consider if using oral contraceptives with the presumption may help bone mass.[40] The parathyroid hormone derivative, teriparatide, has been shown to improve BMD and reduce fractures in adults but has not been used in children due to potential risks of osteosarcoma.

Wnt pathway inhibitors, also referred to as anti-sclerostin antibodies, have a dual mode of action, inhibiting resorption and inducing formation. Results are pending from a clinical trial on the patients with OI treated with romosozumab, an anti-sclerostin antibody (NCT04545554). Anti-transforming growth factor beta therapies are also being considered but are primarily in the preclinical phase.

Summary

Promoting bone health in children is of utmost importance because reaching one's maximal possible PBM during childhood is critical for long-term health. The etiology of pediatric osteoporosis differs markedly from that of osteoporosis in adults. Diagnosis may rely on imaging techniques, but physicians can help identify overlooked patients by incorporating key clinical examination findings, taking focused histories, and obtaining relevant laboratory tests. Furthermore, the orthopaedic physician must maintain a high level of suspicion when evaluating fractures secondary to low-energy mechanisms, fractures of flat bones, or those with fractures that have characteristics unique to certain disorders.

Identifying the child at risk for osteoporosis early will help initiate additional consultations and care specific to that child. With early diagnosis and appropriate

treatment, the pediatric skeleton has the potential to respond and improve BMD. The management of childhood osteoporosis necessitates a multidisciplinary team of health professionals, including orthopaedic surgeons, to enhance disease awareness, mobility, and nutrition and to determine the appropriate course of treatment. Additional research studies on treatment options and their outcomes are essential to improve prevention strategies and enhance the quality of life for a wide range of pediatric bone health conditions.

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