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Pediatric low bone density: when to refer

Abstract

Osteoporosis is a highly prevalent disease-causing high morbidity and health-care expenditures. As bone mass structure rarely varies from that of young adulthood, it is important for early recognition of low bone density disorders during childhood. This article will differentiate the common conditions causing low bone density and help Pediatricians promptly identify the patients in need of a Pediatric Endocrinologist referral by explaining the pathophysiology, evaluation, and management of these conditions.

Keywords: bone fragility, low bone density

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Abbreviations: DEXA, dual-energy X-ray absorptiometry; BMD, bone mineral density; BMC, bone mineral content; aBMD, areal bone mineral density; BMAD, bone mineral apparent density; TBLH, total body less head; VFA, vertebral fracture assessment; CP, cerebral palsy; OI, osteogenesis imperfecta

Introduction

There are multiple medical conditions that predispose to low bone density and fragility fractures. As a primary care provider, it is important to recognize indications for referral to a Pediatric Endocrine bone clinic.

Children are at a greater risk for fractures than adults. Around 42% to 64% of boys and 27% to 40% of girls can potentially develop a fracture between birth and 16 years of age. The upper extremity fractures, especially the forearm, are more common, as more than 65% of all long bone fractures take place in this area.[1]

Osteoporosis is a skeletal condition in which bone density is low and there is increased risk for fractures. A definite indication of osteoporosis is the presence of a vertebral compression fracture without any underlying lesion or impactful trauma. A patient can also be diagnosed with osteoporosis if he/she has a clinically significant fracture history and a bone mineral density (BMD) Z-score less than/equal to -2.0. This can be a result of two or more long bone fractures before 10 years of age or three or more long bone fractures before 19 years of age. Factors such as the location of the injury, imaging features, and the clinical context should all be reviewed when considering a complete pathology evaluation and/or diagnosis of the disease. If choosing to proceed, the primary step is to rule out any evidence of mineral disorders followed by evaluation for any acute or chronic systemic illnesses. If all these tests results are reassuring, then proceed with X-ray spine (with/without skeletal survey) and a dual-energy X-ray absorptiometry (DEXA) scan.[2]

Bone mineral density is a strong indicator of a patient's overall bone health. The dual-energy X-ray absorptiometry (DEXA) scan can report bone mineral content (BMC), areal bone mineral density (aBMD in gm/cm^2), bone mineral apparent density (BMAD). Bone mineral density is reported as Z-scores in pediatric subjects, in which these scores are corrected to the height of the subject. If the Z-score is less than or equal to -2.0 standard deviations, then it is indicative of "low bone mineral mass or bone mineral density". The most frequent areas measured for assessing BMD in children are the posterior-anterior spine and the total body score without including the head. Distal forearm, proximal femur, lateral distal femur, and vertebral fracture assessment can also serve as alternate sites in the case of non-removable artifacts and/or skeletal deformities causing difficulties with positioning. Factors outside of the DEXA score are also equally important criteria in the diagnosis of osteoporosis.[2,3]

As a primary care provider, it is especially important to identify key conditions that can potentially increase the risk for low bone mineral mass. Predisposing conditions such as cerebral palsy, long-term use of steroids, hypogonadism, and certain genetic conditions like osteogenesis imperfecta can negatively affect the subject's overall bone health and deter long-term growth.

The most common condition predisposing to low bone density is cerebral palsy (CP). Calcium intake, medications, and weight-bearing status are the three primary determining factors of bone mass in these patients. Although calcium intake is typically low in these patients, these numbers are not reflected in lab results due to compensatory mechanisms. Medications (such as anticonvulsants, depot medroxyprogesterone acetate, etc.) can deter absorption and/or metabolism of vital nutrients, which has a negative effect on bone health. Bone structures are regulated by simultaneous action of osteoblasts and osteoclast cells; the activities of these cells are highly dependent on the weight-bearing status of the subject. It is shown that children who are non-weight bearing have thinner, more fragile bones than those who can walk.[4] Therefore, these children are inherently at a higher risk for developing fractures (around 4% per year) than those who are weight-bearing. This fracture rate can increase to 7% per year once the child develops a single fracture. Coincidingly as the child's bone health deteriorates, the morbidity rate increases, demonstrating the need for early-recognition of this complication.

Long-term use of steroids can also negatively affect bone health. Effects of long-term use of corticosteroids on skeleton include apoptosis of all bone cells including osteoblasts, osteocytes, and osteoclasts. Osteoblastogenesis is reduced whereas osteoclastogenesis is increased. There is reduction in the synthesis of insulin like growth factor-1 (IGF-1) and collagen. Bone loss can occur in two phases. The initial 10-15% of loss happens in the first few months of steroid use and is followed by phases of 2-5% of bone loss yearly. A daily prednisone dose of ≥7.5 mg for 3 months or more cause significant bone loss and double the risk for fractures. Inhaled steroids on a long term can also potentially affect bone density. Patients who are susceptible for bone loss include those who have elevated parathyroid hormone (PTH), impaired calcium absorption, increased urinary calcium excretion, vitamin D deficiency, and acidosis.[5,6]

Hypogonadism is another condition that can predispose to low bone density. Although estrogen and androgen can both prolong osteoblast and osteocyte survival, they shorten the lifespan of osteoclasts. Evaluation for hypogonadism starts with checking levels of luteinizing hormone, follicle stimulating hormone, testosterone, estradiol. This can be managed by treating the cause and/or replacement therapy with sex steroids, which has shown to improve bone density.[7]

There are certain genetic conditions that can cause low BMD such as osteogenesis imperfecta (OI), WNT1 mutations, etc. The altered collagen formation and deposition in these conditions cause recurrent fractures. Biochemical tests are usually unremarkable. Bone turnover markers are helpful only as a response to treatment and not for diagnosis. Bisphosphonate infusions are usually used as supportive therapy during bone growth. In the case of continued bone pain after reaching maximum bone growth, then bisphosphonates can continue to be used.

Management of low BMD includes ensuring adequate intake of minerals such as calcium, phosphorus, zinc, magnesium, and vitamin D. In those who are non-ambulatory, it is recommended that their serum 25-hydroxy vitamin D level is at least 30 to 40ng/mL. Weight-bearing exercises, especially by physical therapy, are extremely important. Aquatic therapies can also be very beneficial. Pharmacologic management using bisphosphonates are also considered for those with increased bone fragility. Bisphosphonates serve to increase bone mineral density by inactivating osteoclasts, which are responsible for the breakdown of existing bone cells. Bone mineral density typically increases during the period of treatment and then decreases back to baseline within 2 years of discontinuation. Despite this return, a high proportion of children remain fracture free for 5 years or more.

Conclusion

Evaluation of bone health should identify children who may benefit from interventions to decrease their elevated risk of a clinically significant fracture. Indications for referral include a history of multiple fractures and non-weight bearing situations such as cerebral palsy, chronic use of steroids, hypogonadism, and genetic conditions like osteogenesis imperfecta. Early identification and management of these conditions are equally important to reduce both the morbidity of the subject as well as health-care costs.

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Conflicts of interest

The authors declare no conflict of interest.

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